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FLOW AND PRESSURE IN THE ARTERIAL SYSTEM, THEIR HEMODYNAMIC RELATIONSHIP, AND THE PRINCIPLES OF THEIR MEASUREMENT

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BY THE SYSTOLIC ejection of blood into the arterial system, the heart causes a flow of fluid, the rate and course of which depend in the first rank on the diastolic filling and the momentary muscular conditions of the ventricle, and on the arterial pressure. The ejected volume extends the arteries and raises the pressure above its diastolic value. This sudden event initiates a pulse wave in the elastic arteries which is propagated through the system up to its branches where wave reflections take place. At the end of systole, the pressure is usually increased in all arteries, each of them being augmented in volume and sharing in the systolic storage of blood. The subsequent elastic recoil of the arterial walls maintains the blood flow through the capillaries during diastole.

First, the course of blood flow generated by the contraction of the ventricle, and the pressure course in the arteries near the heart, have to be considered. As an example, a selection of tracings obtained by Wetterer and Deppe [49] is seen in Figures 1 to 4. The flow rate in the unopened ascending aorta has been recorded with an electromagnetic flowmeter, the pressure in the lower carotid artery with Broemser's optical manometer,[5] and the femoral pulse with a sphygmograph. The chest of the animals was opened.

In Figure 1, the tracings of pressure and pulse taken on a rabbit show some well-known facts. In each pulse, the pressure rises suddenly, reaches its maximum near the middle of systole, and then decreases towards the end of systole where the incisura appears. During diastole, the pressure is generally falling, but, due to a superimposed

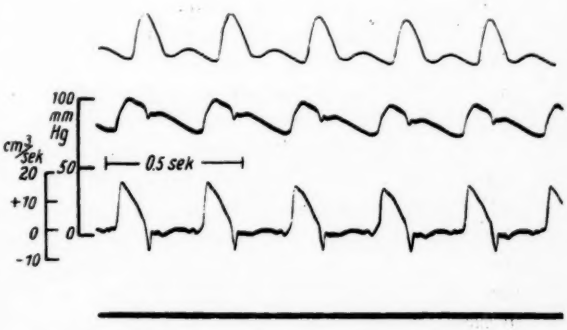

Fig. 1. Records taken on a rabbit. Tracings from bottom to top: flow rate in the ascending aorta, pressure in the carotid, femoral pulse.

wave, there is an elevation in the first half of diastole, synchronous with a deep depression of the peripheral pulse. In this way, the standing wave in the aorta makes its appearance. The flow rate rises suddenly, too, reaches its maximum before the maximum of pressure, then turns back to zero and becomes negative for a very short time at the end of systole during the closure of the aortic valves. During diastole, the flow remains practically at zero.

A large stroke volume was obtained by adrenaline, as demonstrated in Figure 2. In this special case, the flow course is characterized by a plateau or two maxima, respectively. This is a very

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unique phenomenon which was observed in no other animal. In consequence of the high peripheral resistance and the increased pulse wave velocity, the course of pressure is remarkably ele-

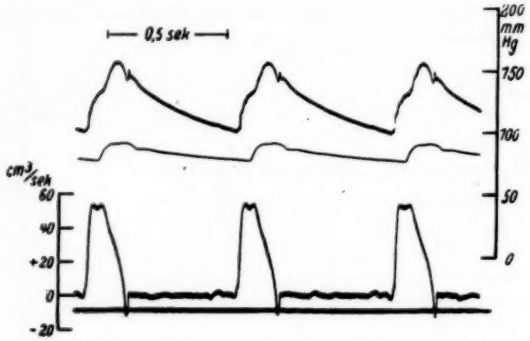

Fig. 2. Records taken on a rabbit after injection of adrenaline. Tracings from bottom to top: flow rate in the ascending aorta, femoral pulse, pressure in the carotid.

vated in the last part of systole by a large reflected wave, the onset of which is clearly seen. The relatively small pressure fall after the systolic maximum and the curved descent of pressure during the prolonged diastole are obvious.

An opposite state of circulation is seen in Figure 3 after the injection of pilocarpine. The peripheral resistance and the mean pressure are low, and the pulse wave velocity is slow. At the end of systole, there is only a minute negativity of flow and, therefore no visible incisura in the pressure curve. After the relatively deep descent from the systolic pressure maximum to the end of systole, the diastolic pressure course is almost horizontal. The time interval between the maxima of flow and pressure is very short. In this case of low pressure level, the courses of pressure and flow are nearly congruent, if presented on the same scale.

Figure 4 shows a record obtained on a dog of 15 kgm. The maximum of flow rate approaches 200 cc. per sec. Attention is to be called to the fact that there are no significant differences in shape of the normal flow course between rabbits, cats, dogs, and monkeys. It may be concluded that the contour of flow course in the human ascending aorta agrees in the principle with those which have been recorded on these animals.

Obviously, the integral of the flow rate (that is the area enclosed by the curve of the flow rate and by the base line of flow) represents the total stroke volume minus that minute part flowing through the coronary arteries during systole. The cardiac ejection curve (that is the time course of

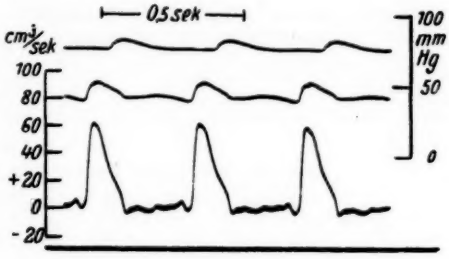

Fig. 3. Records taken on a rabbit after injection of pilocarpine. Tracings as in Figure 1.

volume ejected by the ventricle) results from the stepwise integration of the flow curve from the beginning to the end of ejection time. Some ejection curves, obtained in this way, are seen in Figure 5. Above, the original curves of flow rate. The ejection curves are S-shaped. Generally, the steepest point which corresponds to the maximum of flow rate is situated within the first fourth or third of the ejection time. On the right hand of Figure 5, there is an ejection curve calculated by Remington and Hamilton[40] with their pulse contour method. On the whole, the differential quotient of this calculated ejection curve agrees well with the recorded flow curves although the maximum of the recorded flow course is usually reached somewhat earlier than that of the calculated curve.

Because of the intricate structure of the arterial system, the theoretical treatment of the hemodynamic events is faced with many difficulties. Therefore, several authors tried to formulate the fundamental laws concerning pressure and flow by the study of simplified models. Two different models have proved valuable: the so-called Windkessel and on the other hand, a long elastic tube filled with fluid through which pulsewaves are propagated. The Windkessel model, as shown in Figure 6, consists of a distensible reservoir, representing the distensibility of the total arterial system, an inflow valve representing the aortic valves, and a narrow outlet tube representing the peripheral resistance. Instead of the distensible reservoir, an air-filled rigid box can be used; hence the name air-chamber

or Windkessel. If fluid is injected rhythmically by means of a pump, imitating systole and diastole of the heart, the pressure in the Windkessel rises and decreases rhythmically. In 1899, Frank[10] from the integration of Frank's differential equation under the assumption of a certain flow course. Thus, a slowly ascending S-shaped pressure course results during systole. The diastolic pressure descent describes, as Frank stated, an exponential curve, if the product resistance times distensibility is constant during diastole. In the limits of a normal diastole, the curvature of the diastolic pressure course is only slight so that it approaches a straight line.

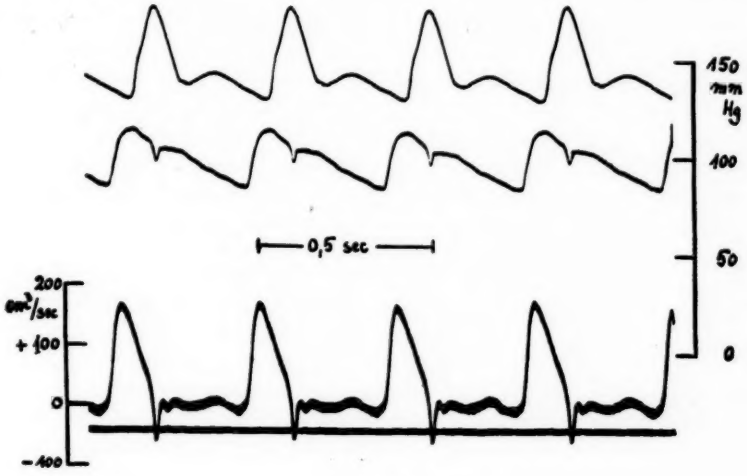

Fig. 4. Records taken on a dog of 15 kgm. Tracings as in Figure 1.

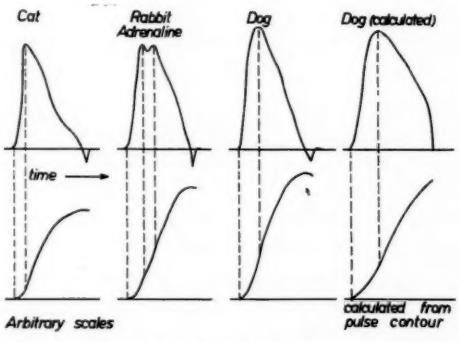

Fig. 5. First, second, and third curve from left to right: Derivation of the cardiac ejection curve (below) from the recorded course of flow rate in the ascending aorta. Last curve at the right hand: Ejection curve (below), calculated by Remington and Hamilton from the pulse contour, and its differential quotient (above).

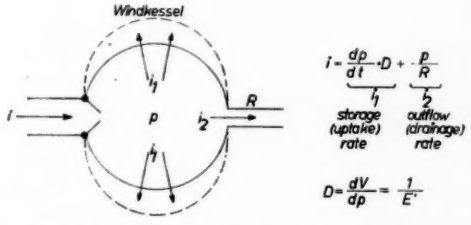

Fig. 6. Design of the simple Windkessel model.

described the relation between rate of inflow i and pressure p by the equation noted in Figure 6 at the right hand. D is the distensibility, that is the ratio of a change in volume to a change in pressure, and R is the resistance. It is seen that the inflow rate is resolved into storage rate i_1 and an outflow rate i_2. i_1 distends the elastic wall and results in an uptake of fluid, while i_2 represents the drainage out of the system.

The pressure course in the Windkessel results

Wetterer[50] emphasized that the systolic pressure course in the Windkessel is quite different from that in the normal arterial system. Nevertheless, the following time correlations between flow and pressure during systole are valid for both the Windkessel and the arteries: the maximum of flow precedes the maximum of pressure; the maximum of pressure precedes the maximum of ejected volume. These relations hold true with regard to the total arterial system as well as to its single branches.

The Windkessel model also permits the prediction of the effects of changes in stroke volume or minute volume, in distensibility or resistance on the mean pressure level and on the pulse pressure. In this regard, Wiggers[61] investigated similar physical models in an experimental way in 1938.

It was mentioned above that the Windkessel model is unable to deliver a systolic pressure course analogous to that of the normal arterial system. However, an extremely high peripheral resistance can give rise to an arterial pressure contour similar to that in the Windkessel, for instance, in cases of renal hypertension as recently published by Remington.[41] But usually, the arterial pressure during systole ascends much steeper, reaches its maximum earlier and decreases, after the maximum, to a greater extent than the pressure in the Windkessel does. In 1940, Wetterer[50] tried to explain these differences between the model and the natural circulation system by the following consideration: In all parts of the Windkessel reservoir the pressure is the same at a certain moment so that the total distensibility is effective during the entire pulse time. But in the arterial system, the distensibility is variable and depends on the distance covered by the pulse wave. In the beginning of the systolic inflow, the pulse wave starts from the aortic root. For instance, 10 milliseconds after, the top of the wave will have passed through about four centimeters. Only the distensibility of this short part of the aorta will be available for the uptake of blood injected during the first 10 milliseconds. The longer the distance covered by the pulse wave, the greater will be this part of the arterial system in which uptake occurs. Consequently, the actual distensibility during systole is variable and a function of time. Wetterer calculated this variable distensibility from the pulse wave velocity and the cross sectional area of the aorta under certain simplified assumptions. Furthermore, he supposed the drainage rate obeying two different laws: first, the simple law of Poiseuille, and second, another relation resulting from the consideration that the drainage rate increases with the number of arterioles reached by the pulse wave. In this way, the flow course was calculated from the pressure course and inversely, and in many cases a satisfactory correspondence between the calculated and the recorded curves was obtained. On the other hand, this calculation was failing in cases of a very high or very low peripheral resistance. Therefore, it may be stated that this extension of the simple Windkessel theory improves our classical model to a certain degree, but cannot take into account all variations occurring in circulation.

Independently of these considerations, Hamilton and Remington[21] developed in 1946 and 1947 a new method of calculating the cardiac ejection curve from the pressure course. The distensibility used in their calculations was not derived from the pulse wave velocity, but from volume pressure relations directly determined on dead animals. The arterial system was divided into six main beds, the individual volume uptakes of which were tabulated at different pressure levels. Also the pulse transmission time to and through each bed was listed at various pressures. Using these statements, the authors computed the time course of the volume uptake from the pulse pressure recorded on living animals. Obviously, the contribution of the separate beds to the momentary uptake varies considerably in the course of systole according to the distance covered by the pulse wave, and the variation of the actual distensibility as a function of time, is a striking feature of this method too. Furthermore, Hamilton and Remington described the systolic drainage of blood as a complex function of the distance covered by the pulse wave and of the arterial pressure. The total ejection curve, resulting from uptake plus drainage, was found to be an S-shaped curve as demonstrated in Figure 5 and also confirmed by cardiometer curves of Wiggers and Katz.[59]

The percental contribution of the various arterial beds to the total distensibility, as it may be quoted from the tables of Hamilton and Remington, is very different. The entire aorta comprises at the most 50 per cent of the total distensibility, the arteries of head, neck, and forelegs nearly 30 per cent, the visceral plus hindlimb arteries about 20 per cent. Of course, also the smallest arteries of each bed contribute to the distensibility.

Wetterer and Pieper[54] developed in 1952, a direct method for determining the total arterial distensibility on living anesthetized animals. A piston pump filled with Ringer solution was connected to the aorta by a tube introduced through the carotid. The piston moved forward and backward and effected rhythmic sinusoidal volume changes; each pump cycle lasted three to five seconds. The volume shift of the piston was adjusted to the size of the experimental animal. In

dogs of 8 to 30 kgm. volume changes of 10 to 30 cc. were used. The blood pressure in the aortic arch, recorded with a miniature manometer,[52,53] increased and decreased with the rhythm of the pump motion. On these relatively slow fluctuations, the arterial pulses were superimposed, as shown in Figure 7. The pulse transmission time through the whole arterial system is exceedingly short compared to the duration of a pump cycle. Therefore, the arterial system reacts to the pump like the uniform reservoir of a simple Windkessel model. For evaluation, the measurements were carried out on the diastolic parts of the pulses. Within a pump cycle of the aortic pressure course, two pulses were selected, one in the ascending part and the other one in the descending part. The mean pressures during the diastoles of both pulses should not differ from each other by more than 2 per cent so that the mean diastolic drainage rates may be supposed to be equal. The distensibility of the total arterial system was given by the formula:

$$D = \frac{i_1 - i_2}{S_1 - S_2}.$$

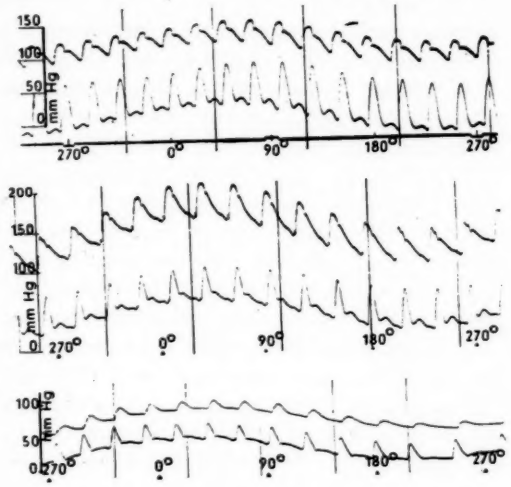

Fig. 7. Tracings obtained on anesthetized dogs during the action of the pump (see text). Above: normal state; aortic pressure and femoral pulse. Middle: high pressure during infusion of adrenaline after atropinisation; aortic pressure and femoral pulse. Below: low pressure due to a ganglionic blocking drug (Pendiomid); femoral pulse and aortic pressure.—Distances between the vertical lines=1 sec.—Numbers below zero-line indicate the angle-degrees corresponding to the sinusoidal movement of the piston. 270°=lowest position, 90°=highest position of the piston.

i_1 and i_2 are the flow rates of the pump during the diastoles of the two pulses, calculated by means of a sine table. S_1 and S_2 are the diastolic pressure slopes of the two pulses. The drainage cancels out in this formula because it is the same for both pulses. The evaluation of ten to twenty pairs of pulses in every record containing several pump cycles gave a standard deviation of about 8 per cent and a mean standard error of less than 3 per cent.

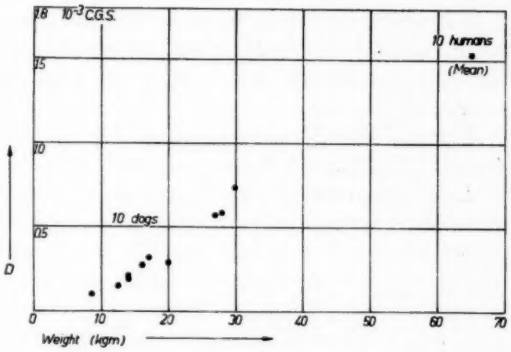

Fig. 8. Total arterial distensibility of ten anesthetized dogs, determined with the pump method. Above at the right hand: Mean of arterial distensibility, determined on ten humans by Warner *et al.*

Figure 8 shows a summarizing diagram of the results obtained on ten dogs of body weights between 8.5 and 30 kgm. in a normal state of circulation. The distensibility, expressed in C.G.S. units, is plotted against the body weight in kgm. The relation between both variables may be regarded as a curve passing through the origin of the co-ordinates and becoming steeper in the range of greater body weights. As far as it can be seen from ten observations, the distensibility is not highly influenced by differences in age and sex of the dogs. The amount of distensibility measured on dogs of 28 to 30 kgm. agrees well with the data calculated from the tables of Hamilton and Remington[21] under the condition of a normal pressure level. Moreover, the results obtained on dogs with the pump method may be substantiated by the investigations of Warner, Swan, Connolly, Tompkins, and Wood[36] who computed on humans, the arterial distensibility from the aortic pressure pulse and from the cardiac output determined by Fick's principle of the dye method. The mean of distensibility determined on ten normal humans and expressed in C.G.S. units

is noted in Figure 8 under the assumption of a mean body weight of 65 kgm. Obviously, an extrapolation of the values measured on dogs points almost directly to the mean of human arterial distensibility.

If the distensibility of the entire human arterial system should be simulated by an air-chamber model, an air-cushion of 1.5 to 2 litres would be required.

Now, the arterial pressure course during diastole may be discussed. It was explained above that in diastole the descending pressure in the simple Windkessel follows an exponential course which represents almost a straight line. In the arterial system, the diastolic pressure course resembles a straight line, too, if the diastole is not abnormally long, and if pressure changes induced by wave reflections do not interfere. At a certain pressure level, the slope of this course is reciprocal to the product distensibility times peripheral resistance. An undisturbed diastolic pressure course may be obtained at the node of the main standing wave in the aorta.

If, in an abnormally prolonged diastole, the pressure falls over a wide range, we find a diastolic pressure course which follows a strongly curved line. Extrapolating this curve beyond the end of diastole, one does not reach values which correspond to the capillary or venous pressure. Rather the extrapolated curve seems to be asymptotic at a level which is far above the capillary pressure. Several reasons explain this fact: First, the arterial distensibility is a function of pressure; it increases when the pressure decreases. Second, the peripheral resistance, too, is a function of pressure because the lumen of the arterioles decreases with falling pressure. Both phenomena result in a rise of the product distensibility times resistance. Hence, the pressure course is flattened. Furthermore, in arterioles and capillaries the relation between pressure and flow of blood does not obey Poiseuille's law because blood deviates from the properties of a homogeneous viscous fluid in narrow vessels. As to the diastolic pressure course, see the publications of Frank,[12] Wiggers,[62] Hamilton,[24] Gomez,[17] and Deppe.[8] The complex relationship between pressure and flow through the arterioles and capillaries has been studied chiefly by Hess,[23] Whittacker and Winton,[58] A. Müller,[32,33] Green and co-workers,[18] Pappenheimer and Maes,[34] Lamport,[28] Rashewsky,[38] and recently by Wezler.[56,57]

In the following, the arterial pulse waves shall be discussed. The theory of waves running through elastic tubes has been founded by the brothers Weber,[47] by Moens,[31] Korteweg, and others during the nineteenth century. Several authors contributed to its further improvement in the twentieth century, chiefly Frank[14] since 1920. Also Apéria,[2] in collaboration with a mathematician, Müntz, and recently Lambossy[27] were working on the wave theory. Landes[29] demonstrated that the fundamental hydromechanical wave equations are analogous to those which are valid for the transmission of electric impulses through telegraph-cables. The mathematical wave theory cannot be given here; the explanations may be restricted to some simplified considerations.

If a certain amount of fluid is pumped quickly into an elastic tube containing fluid, the whole column in the tube resists to a sudden dislocation due to its inertia. First of all, the wall of the initial part of the tube is distended by the injected volume. Then the elastic force of the extended wall causes a shift of fluid to the next segment, and so on. Therefore, the extension is propagated along the tube and is accompanied by a pressure rise and a movement of fluid from one segment to the next one. If there is no wave reflection, the courses of wave-pressure and wave-flow will be in phase. This is very important for the understanding of the contour of certain pressure pulses. The ratio of pressure to flow rate of the wave is a sort of impedance and may be called the wave-resistance R_w:

$$R_w = \frac{\delta \cdot c}{A}.$$

δ is the density of the fluid, c the wave velocity, and A the cross sectional area of the tube. Friction and viscosity are not considered in this formula. Another formula developed by Frank[14] in 1920, and by Bramwell and Hill[4] in 1922, shows the direct relation between the wave velocity, the density and the so-called volume elasticity modulus α. The latter represents the reciprocal of the distensibility per unit volume.

$$\alpha = \delta c^2.$$

If the tube wall effects a viscous resistance besides its elasticity, the volume elasticity modulus and the pulse wave velocity depend to a certain extent on the quickness of stretching. Concerning the arteries, Ranke [37] investigated this phenomenon in

a theoretical and experimental way. It gives rise to effects of hysteresis, particularly in the first part of systole.

The formula of Frank and of Bramwell and Hill has been used, and also discussed by many investigators, because it renders the possibility to calculate the arterial distensibility from the pulse wave velocity. The application of this formula postulates the measurement not only of the wave velocity, but also that of the total tube volume V or its equivalent:

$$D = \frac{V}{\alpha} = \frac{V}{\delta c^2}$$

The measurement of V is difficult in the case of arteries *in situ* because the arterial diameter is not the same in all sections and depends on the pressure and on the vasomotor tone of the muscular vessels.

In order to obtain an equivalent of volume V, Frank and his associates multiplied the cross sectional area of the ascending aorta by a certain length. Broemser and Ranke[6] supposed that this length was represented by the distance covered by the pulse wave during the ejection time. Frank[15] used the half of the wave length of the main standing wave, Wezler and Böger[55] only one-fourth of this wave length. Bazett and co-workers[3] tried to improve the method by a more detailed consideration of central and peripheral arteries. On the average and under normal circulatory conditions, the values of distensibility, obtained on dogs by Frank's calculation, agree with those determined by the pump method of Wetterer and Pieper[54] which has been described above. The values calculated with the method of Broemser and Ranke are similar or somewhat larger; those of Wezler and Böger are smaller than Frank's data. The calculations of the arterial distensibility from the pulse wave velocity were used for the estimation of the stroke volume in different ways, the validity of which has been examined by many comparison experiments. In numerous cases a satisfactory agreement with the results of the comparison methods was observed; in other cases, the formulas gave remarkable deviations. This is not surprising because, up to now, there is no hemodynamic calculation which respects all the variable properties of the arterial system. Not only the calculation of the arterial distensibility from the wave velocity involves certain difficulties, but also the determination of the ratio of systolic to diastolic drainage may imply some errors, particularly in cases of high pulse pressures and a high arteriolar vasomotor tone.

The pulse contour method of Hamilton and Remington[21] is based on more experimental data than on indirect calculations. Its fundamental conception consists chiefly in the direct and detailed measurement of the arterial distensibility and in the consideration of the pressure distribution in the arteries at the end of systole. About the latest development and the range of applicability of this method see the paper of Dr. Remington in this volume.

Reflections of tube waves occur when the wave resistance, that is the quotient c/A, is changing from one tube segment to another. Generally, this quotient increases from the heart to the periphery so that we conclude that wave reflections take place within the whole arterial system, especially in the arms and legs. Since the increase in wave resistance is highest in the terminal arteries, the major part of all reflections must occur here. It is obvious that the vasomotor state of these vessels determines the amplitudes of the reflected waves.

In his interesting conception, Remington[41] presented the arterial system as to be fused into a single funnel-shaped tube. The length co-ordinate of this figure corresponds to the pulse transmission time, and its local diameter to the distensibility per unit transmission time. It can be demonstrated by a simple calculation that this diameter is inversely proportional to the wave-resistance.

Reflected waves running to the aortic root are reflected anew. The interference of the primary pulse wave with reflected waves, or of reflected waves with one another, gives rise to standing waves which have been described first by Frank.[12,15] The main standing wave, thoroughly investigated by Hamilton and Dow,[20] is generated between the heart and the leg arteries. It is characterized by periodical damped undulations which are superimposed on the fundamental pressure contour. In the arteries near the heart, the phase of these undulations is opposite to that in the lower aorta and its branches. Between both regions of opposite phase we find a node which is situated in the thoracic or upper abdominal aorta. The main standing wave comprises half a wave length,[15] or, in other words, the natural period of the standing wave is twice the pulse transmission time from the heart to the peripheral end of the standing wave. Furthermore, the pulse transmis-

sion time from the heart to the node equals that from the node to the peripheral end.[42] In man, the node is usually situated in the upper abdominal aorta[42,24] and can be shifted towards the heart by compression of the leg arteries below or above the knees.[25]

Recently Alexander[1] assumed the standing wave in the aorta to be influenced by a negative wave reflection at the level of the diaphragm where large arteries are branching off from the aorta.

The pressure amplitudes of reflected waves increase if the peripheral resistance is raised, whereas the reflections can be minimized by an extreme vasodilation. The shape of the central pressure pulse is highly influenced, not only by the cardiac ejection course and the drainage, but also by reflected waves, as shown in the Figures 1 to 4. In the state of an increased peripheral resistance and a high pressure level, the pressure will not descend in the second half of systole, but remain at a plateau or even arise to a late maximum because of the superposition of reflected waves. On the other hand, a low peripheral resistance effects a pressure fall from the early systolic peak to the incisura so that the systolic pressure course is similar to the flow course in the ascending aorta. In case of the first extreme, the arterial system approaches the simple Windkessel because the high pulse wave velocity and the multiple and pronounced reflected waves effect an almost uniform filling. The second extreme, however, is characterized by insignificant wave reflections so that the arterial system approaches the simple long tube model in which the course of pressure corresponds to the course of flow. It is seen that the properties of the normal arterial system are intermediate between both extremes and that a combination of the Windkessel and the wave theory is required for the complete understanding of hemodynamics.

The pressure contour in peripheral arteries, for instance in the leg arteries, is generally characterized by a steep and long systolic upstroke, by an increased pulse pressure, and by one or two distinct diastolic undulations, called dicrotic waves, which are related to the standing waves. The shape of upstroke is chiefly caused by the facts that the peak of the pulse wave is propagated more quickly than the foot, and that steady wave reflections raise the pulse pressure. However, the mean pressure is almost the same in all large and middle arteries, provided that the body is in a horizontal position. The main pressure drop takes place in the arterioles.

Within the limits of this brief survey, it is not possible to describe all the details of the pressure course and their changes along the arterial system which have been investigated by many authors, chiefly by Frank and his associates, by Wiggers, by Hamilton, and recently by Laszt and Müller.[30] The so-called water hammer pulse has been analyzed by Alexander[1] and by Peterson.[35] Further particulars concerning pressure and flow may be mentioned in the following methodical notes:

The principles of modern membrane manometers, established by Frank[11] since 1903, are well known. The natural frequency depends on the volume elasticity coefficient of the membrane and on the effective mass of the fluid in the connecting tubes. Well approved optical manometers have been designed by Frank,[13] Broemser,[5] Wiggers,[60] Hamilton.[19] Electrical manometers are advantageous in several respects. Their sensitivity can be adjusted over a wide range only by change in amplification, and the manometers may be operated independently of the distance from the recording camera. The deformations of the membrane can be transduced into electrical magnitudes by changing the electric resistance of a stretched wire, by changing the capacitance of a condensor or the inductance of an electric choke. Also the variable coupling of the two sections of a differential-transformer may be used. Another way of transducing mechanic into electric magnitudes consists in the photo-electric effect. Furthermore, special electronic tubes have been developed in which an internal electrode can be shifted from outside by the manometer membrane.

Special technical problems arose by the cardiac catheterization first performed on man by Forssmann and worked out for clinical use by Cournand.[7] A long and narrow catheter attached to the manometer lowers the natural frequency of the recording system. Furthermore, the catheter becomes distensible if warmed up to body temperature. A suitable damping ratio is required if distortions of the tracings shall be avoided. Hansen[22] published a theoretical treatise about these problems. After Wood,[63] the artefracts caused by impacts on the catheter in the beating heart may be reduced sufficiently by a damping ratio which gives the manometer system a uniform dynamic response up to 10 cycles per second and a sharp

cutoff in sensitivity above this range. A manometer adjusted in this way is able to record the practically important components of the human pressure pluse; but, of course, the particulars of higher frequencies will not be reproduced.

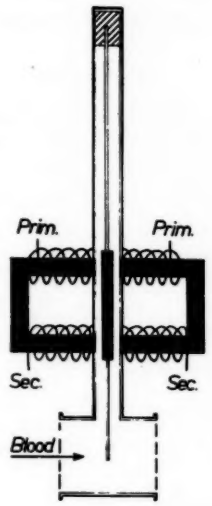

Fig. 9. Sketch of the hydrometric pendulum developed by Pieper and Wetterer. The short horizontal tube is inserted into a blood vessel. The pendulum itself is suspended elastically in the side tube and carries a small slug of iron which, in case of deflection, changes the mutual inductance of the differential-transformer. The natural frequency of the pendulum is more than 200 cps.

A natural frequency of 500 to 1,000 cps, and an artefact-free performance are obtained by miniature manometers attached to the tip of a cardiac catheter. Former devices have been published by Schütz[43] and by Wagner.[45] Wetterer[52] adopted the principle of recording small displacements with the help of differential-transformers for the construction of a miniature manometer with a high natural frequency. This instrument has been improved and adapted for use in humans by Gauer and Gienapp.[16] Ellis, Gauer, and Wood[9] examined its properties in comparison with a strain gauge type.

The measurement of blood flow involves greater difficulties than that of pressure. The mean rate of flow may be measured by instruments called "Stromuhren." Some of them are based on the principle of Ludwig's Stromuhr or on Shipley's[44] rotameter type, and are inserted into an opened vessel while Rein's Thermostromuhr[39] is applicable to unopened vessels. Another sort of flowmeters is able to record the pulsatile flow with all its instantaneous variations. This kind, based on hydromechanic or electric principles, may be called "tachographs." The hydromechanical devices are founded on friction and inertia of the moving blood. For instance, a pressure drop is effected when the blood is led in a Venturi-tube from a wide to a narrow segment. This pressure drop, recorded with a differential-manometer, equals the sum of a term proportional to the blood velocity and another term proportional to the square of velocity. A similar effect is obtained by the Pitot-tubes and their variations. The so-called hydrometric pendulum consists of a minute lever which is suspended elastically in a right angle to the direction of blood flow, and is deflected by the moving blood. In the electromagnetic flowmeter [Kolin 1936;[26] Wetterer 1937[48]], the blood moves through a magnetic field so that potential difference is induced which is proportional to the blood velocity. The application of an alternating magnetic field, introduced by Kolin, has added considerably to the further improvement of this method.

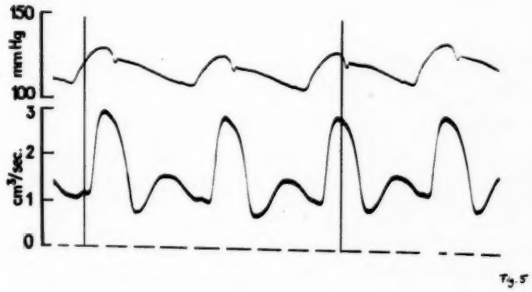

Fig. 10. Course of flow rate in the femoral artery of a dog, recorded with the device of Fig. 9. Above the pressure course in the aortic arch.

Finally, an improved model of the hydrometric pendulum, recently developed by Pieper and Wetterer,[36] may be demonstrated. The deflections of the pendulum are transduced into electrical magnitudes by means of a special differential-transformer as shown in Figure 9.

The course of flow rate in the femoral artery of a dog, recorded with this device, is seen in Figure 10. There is a relatively high diastolic flow rate and a diastolic flow wave which is related to the standing wave.

Figure 11 shows the normal flow course in the carotid. It contains the incisura, coincident with a decline between two maxima. The maximum

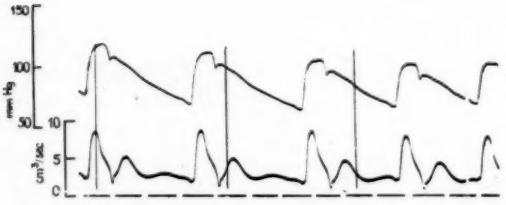

Fig. 11. Normal flow course in the carotid of a dog, recorded with the device of Fig. 9. Above the pressure course in the aortic arch.

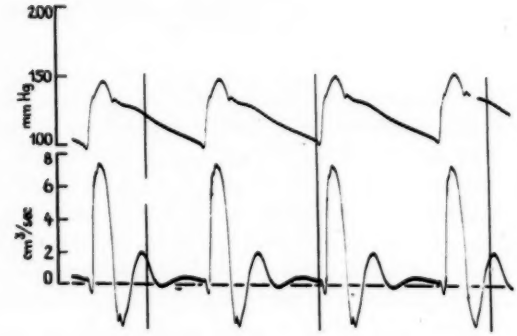

Fig. 12. Caritod flow course after injection of adrenaline. Above the pressure in the aortic arch.

of carotid flow precedes the maximum of pressure in the aortic arch. The diastolic undulations of flow are probably caused by pressure undulations of the main standing wave. But these undulations of flow are much more pronounced than those of pressure. Towards the end of diastole, the flow rate decreases almost corresponding to the diastolic pressure descent.

The effect of a moderate dose of adrenaline is seen in Figure 12. In contradistinction to the previous figure, the mean diastolic flow rate is very low. It must be concluded that, under the condition of an increased peripheral resistance, the main portion of drainage takes place during systole; for the high pressure during systole has a much stronger opening effect upon the constricted arterioles than the lower pressure during diastole.

In the present state of hemodynamics, we are not able to explain all the details concerning the relations between pressure and flow in a quantitative way. But we hope that by improvement of flowmeters we will be able to record pressure and flow simultaneously in many parts of the arterial system in order to obtain better understanding of their interrelationship.

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(Continued on Page 104)

MEASUREMENT OF PRESSURES BY CARDIAC CATHETERS IN MAN

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THE RECORDING of pressures from the chambers and vessels of the right side of heart in man must usually be done by means of a cardiac catheter connected to a manometer. The dynamic response of such a system can quite easily be made adequate for sufficiently accurate reproduction for most purposes of the pressure pulses in question.[1,5,8] However, serious pressure artifacts, caused by impacts on and motion of the catheter imparted by the heart beat, practically preclude high fidelity pressure pulse recording by this method if conventional catheter-manometer systems are used.[1,4] This study was carried out to obtain data concerning the optimal dynamic response characteristics of cardiac catheter-manometer systems for recording of pressure pulses by venous catheterization in man.

Methods

Pressure pulses, and other physiologic variables were recorded during routine diagnostic cardiac-catheterization procedures,[7] by a photo-oscillographic assembly described elsewhere.[6] In the majority of the experiments, pressures were recorded via the catheter by means of a specially adapted strain gauge manometer.[7] The over-all dynamic response of this catheter-manometer system could be varied instantaneously by a multipole switch, connected so as to allow recording of the pressure pulses interchangeably by three different galvanometers with natural frequencies of 5, 12, and 25 cycles per second, respectively. In part of the experiments the catheter pressures were recorded interchangeably, by turning a stopcock either by a high-frequency capacitance manometer,* or the strain gauge manometer. The resonant frequency and damping characteristics of the catheter-manometer assemblies used were determined at the conclusion of each catheterization procedure by recording the responses of the systems to square wave and variable frequency sine wave pressure variations generated by an electromagnetic hydraulic pressure oscillator described elsewhere.[3,6] Care was taken that the conditions of fluid filling and hydraulic connections between catheter and manometer were identical to the conditions pertaining during the experiment.

The susceptibility of the catheter-manometer systems to pressure artifacts, caused by motion of the catheter, was also studied at the termination of each procedure soon after withdrawal of the catheter tip from the vein. The apparatus used consists of a motor-driven cam, which imparts either a sine wave or a square wave motion to a shaft, to which the tip or shaft of the catheter can be clamped. The sine wave motions were at a frequency of 2 per second and peak-to-peak amplitude of 2 cm. The frequency of the square wave impacts was 1 per second. The pressure artifacts, generated when the catheter was subjected to these motions along the axis of the tip and perpendicular to the tip or the midshaft of the catheter, were studied.

Results

Studies of this type have been carried out during thirty catheterization procedures using strain gauge manometer systems only, and in seventeen procedures using the strain gauge and capacitance manometers interchangeably. The catheters used were of the Cournand type, either 100 or 120 cm. in length, and varying in diameter from size 4 to 7 French.

In general, the dynamic response characteristics of catheters of a given size and length, connected to the strain gauge manometer, were quite closely reproducible from procedure to procedure. This reproducibility was not obtained with the higher frequency capacitance manometer system, in spite of extreme care in attempting to insure minimal compliance of hydraulic connections, and

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*The Lilly Manometer manufactured by the Technitrol Engineering Company, 2751 North Fourth Street, Philadelphia 33, Pennsylvania.

avoidance of entrapment of minute air bubbles.

Catheter-manometer systems with low dynamic response characteristics uniformly produced pressure pulse recordings with less evident distortion by motion artifact than did the higher frequency systems. This difference was especially evident for pressure recordings taken when the catheter was threaded through the beating heart, as is the case for pulmonary-artery wedge, pulmonary arteries and right ventricle pressures.

There was an excellent correlation between the degree of distortion of recorded pressure pulses by motion artifacts, and the amount of pressure artifact caused by sine wave, and impact catheter motions produced by the motor-driven cam apparatus. Figures 1, 2 and 3 show typical results illustrating these findings.

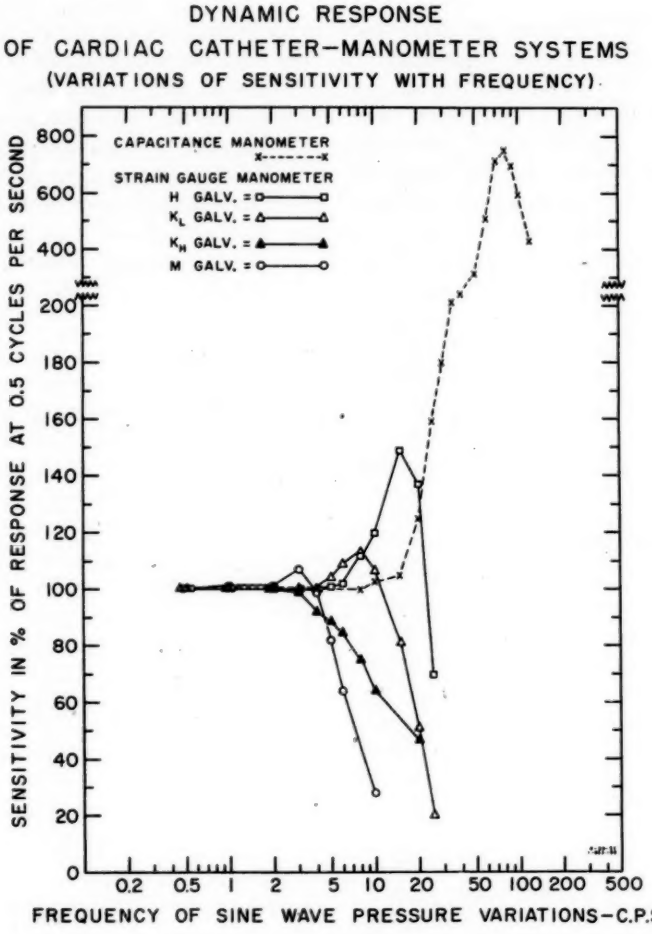

Fig. 1. Variations of sensitivity of cardiac catheter-manometer systems with the frequency of sine wave pressure variations determined immediately after withdrawal of the No. 5 French catheter, 100 cm. long, from the vein (see text for details). The catheter could be connected interchangeably to either a capacitance or a strain gauge manometer. The dynamic response of the strain gauge could be altered interchangeably by connecting it to any one of four different recording galvanometer systems designated as H, K_L, K_H and M.

These studies were carried out during and after a cardiac catheterization of a six-year-old girl found to have pulmonic stenosis. Pressures

were recorded via a 100 cm. No. 5 cardiac catheter from the pulmonary-artery wedge, pulmonary artery, and right ventricle by a strain gauge manometer coupled interchangeably to each of four different galvanometer systems, and by a high-frequency capacitance manometer.

The natural frequency of the capacitance manometer system was 90 cycles per second, and that of the strain gauge-catheter system about 20 cycles per second. By use of lower frequency galvanometers the response could be reduced interchangeably to about 10 or to less than 5 cycles per second (Fig. 1). The susceptibility of these catheter-manometer systems to pressure artifacts caused by motion of the catheter is shown in Figure 2. Since the major portion of the pressure artifacts are caused by the acceleration and deceleration of the fluid column within the catheter, induced by motion of the catheter, it is to be expected that motions along the axis of the catheter generate greater reactive pressures than do motions perpendicular to the axis. Impacts along the axis of the catheter tip caused spikes of pressure varying from 5 mm. of mercury for the overdamped 5-cycle strain gauge system to more than 200 mm. of mercury for the underdamped 90-cycle capacitance manometer system. Sine wave motions at 2 per second of 2-cm. amplitude caused smooth 10 mm. of mercury peak-to-peak pressure variations in the 5-cycle system and continuous noise, 10 to 20 mm. of mercury in amplitude, in the 90-cycle capacitance system. Higher frequency catheter-manometer systems were uniformly more susceptible to motion artifacts than the low-frequency systems.

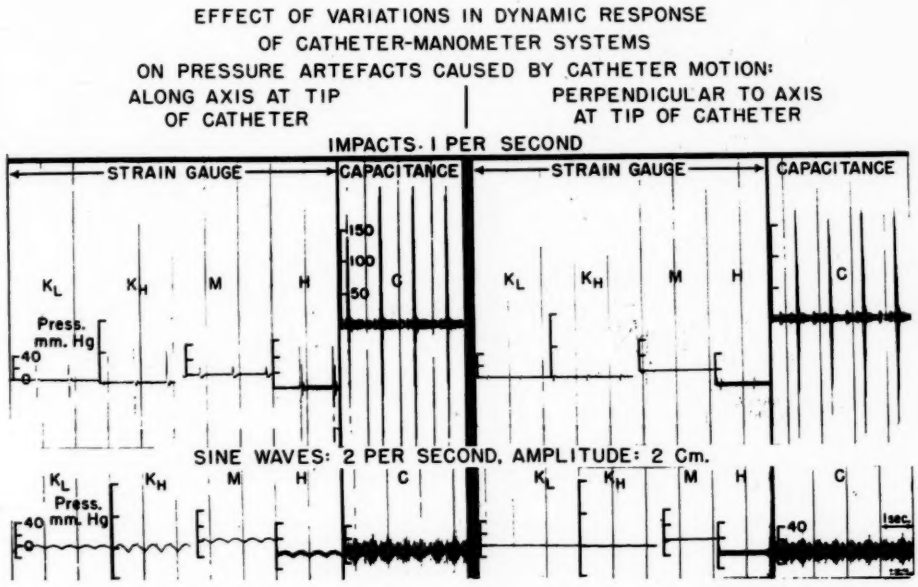

Fig. 2. Effect of variations in dynamic response of cardiac catheter-manometer systems on the pressure artifacts, caused by catheter motion along the axis of the tip of the catheter (left panels), and perpendicular to the axis at the tip of the catheter (right panels). The catheter motions were produced by a motor-driven cam shaft. The dynamic responses of the strain gauge and capacitance manometer systems are shown in Figure 1. Note that the lower frequency strain gauge systems (K_H and M) record less artifact than the 20-cycle system (H), and that the high-frequency undamped capacitance manometer system records continuous high-frequency oscillations probably associated with induced mechanical vibrations in the catheter system. Note also that motions along the axis of the tip of the catheter produce greater pressure oscillations than do identical motions perpendicular to the axis of the catheter tip.

Pressures recorded by these identical catheter-manometer systems from the pulmonary-artery wedge, pulmonary artery and right ventricle are shown in Figure 3. The most artifact-free tracings were obtained by the 5-cycle system. When the catheter was threaded through the heart into the wedge position, the pressure pulses recorded

by the 90-cycle capacitance manometer system were obscured by artifacts.

Tracings obtained by the capacitance manometer can be improved by damping so that the response is limited to 5, 10 or 20 cycles per second. The capacitance and strain gauge systems are then about equally sensitive to motion artifacts. Pressure pulse tracings obtained from the pulmonary artery and right ventricle show the least artifact for the 5-cycle system, most for the 20 and intermediate, and about equal artifact for the 10-cycle capacitance and strain gauge systems.

It has been suggested that motion artifacts would be reduced if very small catheters were used or, better still, if pressures were recorded by a small catheter threaded through the standard size cardiac catheter. In our laboratory this has been found not to be the case. The small catheter used was of the Peterson type (145 cm. long, 0.3 mm. I.D., 0.6 mm. O.D.). For pressure recording from the heart it was threaded through a conventional 120 cm. Cournand No. 6 cardiac catheter using a special adapter assembly.

The dynamic response of these systems was determined. The natural frequency of the 0.3 mm. catheter-capacitance system was about 30 cycles per second. For pressure pulse recording the system was damped down to 10 cycles per second, similarly to the standard catheter-strain gauge system. The strain gauge system, recording from

EFFECT OF VARIATIONS IN DYNAMIC RESPONSE
OF CATHETER–MANOMETER SYSTEMS
ON PRESSURES TRANSMITTED VIA NO. 5, 100 Cm. CATHETER
(Female, 6 yr., Pulmonic Stenosis)

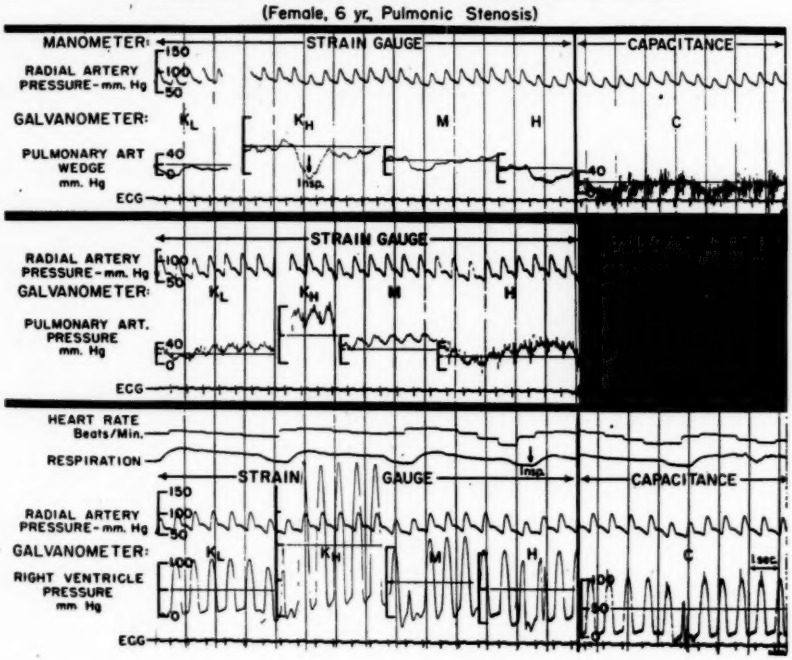

Fig. 3. Effects of variations in dynamic response of catheter-manometer systems on pressure pulses recorded from the pulmonary-artery wedge, pulmonary artery and right ventricle of a six-year-old girl with valvular pulmonic stenosis. The dynamic responses of the strain gauge and capacitance manometer systems are shown in Figure 1. Note that recordings from the lower frequency strain gauge systems (K_H and M), show less evident distortion by artifact than the 20-cycle system (H), and that when the catheter is threaded through the beating heart the pressure pulses in the capacitance manometer recording are almost completely obscured by high-frequency artifact. Note also the correlation between the susceptibility of the various systems to motion artifact shown in Figure 2, and the evident artifact in the pressure pulses recorded by these same manometer systems.

the cardiac catheter containing the small catheter, was the most overdamped of the three systems, having a uniform response out to only 2 cycles per second.

tems with a sharp cutoff in sensitivity to higher frequencies which thus selectively discriminate against the relatively higher frequency motion artifacts. This requirement is, however, mutually

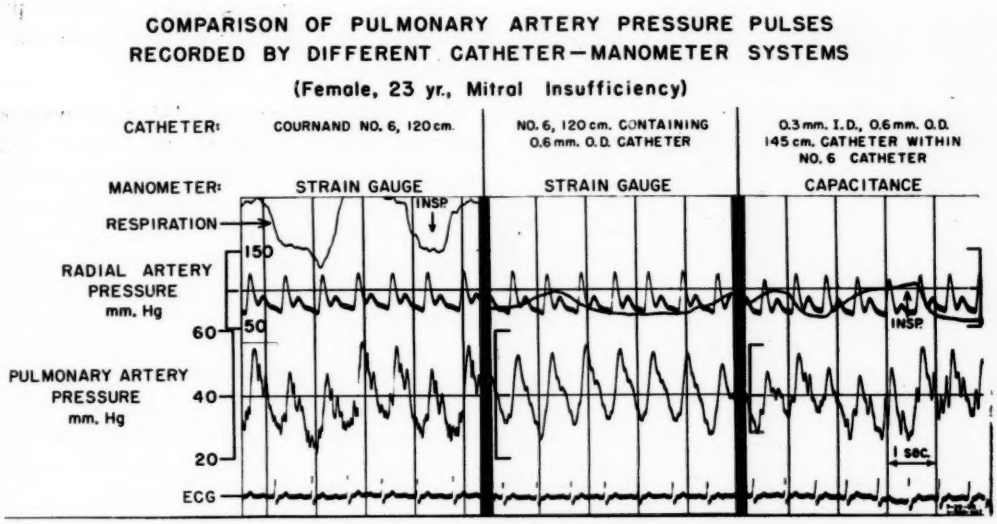

Fig. 4. Comparison of pulmonary-artery pressure pulses recorded by different catheter-manometer systems from a twenty-three-year-old woman with mitral insufficiency. For details concerning the make-up and the dynamic responses and susceptibility to motion artifacts of these three manometer systems, see text. The small plastic catheter was protected from impacts due to the heart action by the No. 6 cardiac catheter through which it was threaded into the pulmonary artery. The pressure pulses recorded by this system (right panel) are nevertheless, still badly distorted by artifact, although somewhat less so than the recordings from the conventional catheter-strain gauge system shown on the left. The frequency response of the overdamped system (strain gauge connected to the No. 6 cardiac catheter containing the small plastic catheter) is probably too low for accurate recording of intracardiac pressure pulses, though the tracings obtained (middle panel) show the least evident distortion.

The susceptibility of these systems to motion artifacts was determined also. The overdamped strain gauge system exhibits the smoothest tracing while the small catheter-capacitance manometer system is intermediate between that and the standard catheter-strain gauge system. Actual pressure pulse tracings from the pulmonary artery of the same patient show similar results (Fig. 4), evident motion artifacts on the small catheter-capacitance tracing being intermediate between those seen on the tracings from the two strain gauge-catheter systems.

Comment

These and similar studies have led us to conclude that it is highly improbable that high-fidelity pressure pulse tracings can be recorded via conventional catheters threaded into the beating heart. The degree of evident distortion can be reduced by using relatively low-frequency recording sys-

antagonistic to the recording of cardiac pressure pulses, since elimination of evident motion artifact requires such a low frequency system that adequate reproduction of the pressure pulses in question may not be possible. Indeed, motions of the catheter at the frequency of the heart beat, induce pressure artifacts at the frequency of the cardiac cycle which may be relatively smooth in contour and fused with the actual pressure pulse in such a way as to be unrecognizable as artifact.

The only solution to this problem known to us at present, is the use of a miniature manometer attached to the intracardiac tip of the catheter. Such a manometer has been made by Gauer and Geinapp after Wetterer's design.[2] Since its moving element has a mass of only 15 mg., reactive forces to acceleration and deceleration of the catheter tip are very small and hence this catheter-manometer system is practically free of motion artifacts when the catheter is moved outside the

body.[1] Pressure pulses recorded from the heart and great vessels are similarly practically devoid of artifact.[1]

Summary and Conclusions

An electromagnetic hydraulic pressure oscillator has been used to study the dynamic response characteristics, and a motor-driven cam apparatus to study the pressure fluctuations caused by reproducible sine wave and square wave motions of cardiac catheters connected to strain gauge or capacitance manometer systems. These studies were carried out immediately after use of these identical systems for recording of intracardiac pressure pulses during forty-seven diagnostic cardiac-catheterization procedures in patients with various types of cardiovascular abnormalities.

1. Sine wave motions, along the axis of the tip of the catheter at a frequency of 2 per second and a peak-to-peak amplitude of 2 cm., produce sine wave variations of pressure at this frequency and with a peak-to-peak amplitude of about 10 mm. of mercury. This motion of the catheter tip resembles that usually seen to some degree at cardiac catheterization, especially when the catheter tip is in the pulmonary artery. Sine wave motion perpendicular to the axis of the tip of the catheter did not produce pressure fluctuations of practical significance.

2. Square wave motions (impacts) produce high-frequency pressure variations of greatest magnitude when the impact is directed along the axis of the tip of the catheter. The amplitude of the recorded pressure variations induced by square wave motion of the catheter (impacts) varied inversely with the frequency response of the catheter-manometer systems used. Impacts of the catheter produced much less pressure fluctuation when in a direction perpendicular to the axis of the catheter than when directed along the axis of the catheter.

3. A close correlation was demonstrated between the susceptibility of cardiac catheter-manometer systems, to pressure artifacts induced by motion of the catheter outside of the body, and the degree of distortion by artifact of pressure pulses recorded by the same catheter system from the beating heart.

4. Catheter-manometer systems, with a uniform dynamic response out to 5 to 10 cycles per second with a sharp cutoff in sensitivity to higher frequencies, were least susceptible to the higher frequency pressure artifacts induced by catheter motion, and likewise produced pressure pulse recordings with the least evident distortion by artifacts.

5. Pressure pulses, recorded by conventional catheters threaded through the beating heart, should be regarded with a high index of suspicion, since the lower frequency artifact induced by motion of the catheter synchronous with the heart beat, may be of such character and be fused with each pressure pulse in such a manner as to be unrecognizable as artifact.

6. Recording of pressures by a small (0.3 mm. I.D.) catheter threaded through a conventional cardiac catheter reduces slightly but not to a sufficient degree the artifact present in pressure pulse recordings from within the heart.

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MATHEMATICAL CONSIDERATIONS OF INDICATOR DILUTION TECHNIQUES

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THE STUDY of circulatory mixing processes is an important practical approach to the investigation of the circulatory physiology of man in health and disease. Injection of substances into the blood stream and observing the time relations of concentration either at the injection site or at other sites is one of the physiological manipulations which can be sanctioned in human studies. With the advent of a growing multitude of tracer substances, there remains an increasing number of experiments of this type which can be done. Their interpretation will require an improved knowledge of the processes of circulatory transport and mixing.

The measurement of volumes of dilution of injected substances will not be emphasized in this discussion. Instead we will consider the immediate events following the rapid injection of a small portion of labeled material into a large vessel. The extensive pioneer work by Hamilton and his associates[5] on the concentration of dye in blood from the radial artery following injection in the cubital vein is familiar to all. Recent developments include the use of radioactive isotopes[1,8,12] and the development of photoelectric recording methods either with the use of the ear oximeter or with the recording cuvette through which blood from a needle in an artery or vein is allowed to flow.[8,10,11]

Although these technical improvements have been of great value, the interpretation of the data which they furnish requires the use of a few mathematical considerations in achieving a quantitative description of both the dispersing processses which occur as the dye flows through the circulatory labyrinth and the effects of recirculation. The present discussion should be considered as an introduction to a problem which has been explored in a preliminary way only, but steady progress is being made and the results seem to justify the effort, at least, so far. We will confine our attention for the moment to substances such as P^{32}-labeled erythrocytes or T-1824 which do not rapidly disappear from the circulation, since it has been shown recently that even rather rapidly disappearing substances yield results which can be expressed in terms of an equivalent nondisappearing substance.[13]

Circulatory mixing represents one of the powerful processes by which the body resists the action of external disturbances which might interfere with the preservation of a stable internal milieu. When a small quantity of injected substance moves through the circulation, we can imagine it to be resolved into a very large number of small elements, each requiring a different amount of time to move through the circulatory labyrinth. The variation in traversal times causes the substance to be randomized and progressively smeared or dispersed in time during the process of circulatory mixing. It is intriguing, perhaps, to realize that here the body architecture achieves design essentially by lack of it. Because of this apparent lack of design, as we usually think of it, the purely deterministic approach is not likely to be fruitful in describing the dispersion process.

One illustration of our limitations, which was particularly instructive, occurred when an attempt was made to investigate the simple case of dispersion of dye during simple laminar flow along a uniform cylindrical tube. The theory of this process is quite simple. Suppose that water is flowing through the tube at the rate of Q cc. per second and that I milligrams of dye are injected in such a way that the initial distribution in the tube is that of a uniform concentration in a thin layer whose plane is perpendicular to the axis of the tube. If the volume of the tube from the injection point to the outflow is V cc., then the concentration of dye (mg./cc.) in any small uniformly mixed sample taken at time t from the outflow will be

$$C(t)\,dt = 0 \text{ for } t < V/2Q \quad \text{and}$$

$$C(t)\,dt = I\,V\,dt/2Q^2\,t^2 \text{ for } t > V/2Q$$

(Equation 1)

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Figure 1 shows a comparison of the results of one attempted experiment with the theoretical prediction. The results do not agree in anything but a qualitative sense. Further investigation showed that it was extremely difficult to establish the required initial distribution. Not only did small differences in specific gravity of the injected dye compared to the water in the tube cause settling of the dye, but also the concentration was not uniform because of the slow mixing of the dye with the water. Since the arrival time downstream of a small dye element depends critically on its initial radius from the center of the tube, the time relations downstream were greatly affected by the nonuniformity of the initial dye distribution, and due to our inability to control the dye, the results were erratic from one experiment to the next. Although this difficulty may be controlled by special techniques, the difficulty in control of the dye applies in all likelihood to a great many typical physiological situations.

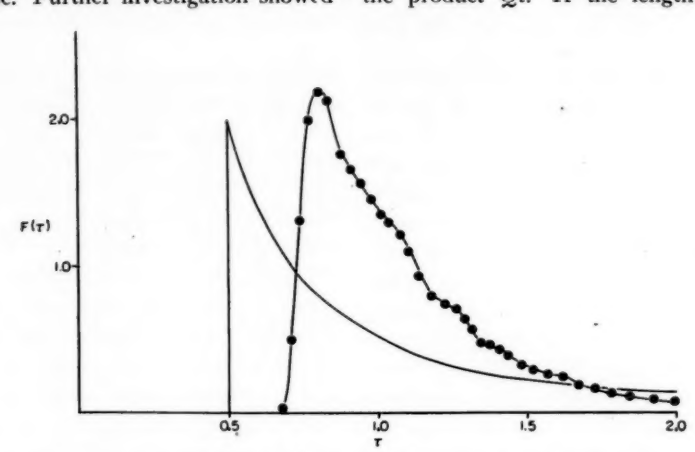

Fig. 1. Theoretical and attempted experimental results on the outflow of a small uniformly injected element of dye from a uniform cylindrical tube in which the transporting fluid moves without turbulence. The experimental results (dotted points) fail to confirm the theory because of uniform inability to control initial distribution of injected dye and to prevent gravitational settling.

Equation 1 can be greatly simplified if we choose a different set of variables. In the first place the concentration in a given sample is not the basic quantity but rather the fraction of the total injected dye contained in it. Secondly, the flow rate, the time, and the bed volume are not individually important variables. Neglecting diffusion and settling out effects, in the ideal situation we can collect the fractions at any rate, or at variable rates, or the experiment can be interrupted and continued at a later time. The factor controlling the situation at the outflow is the volume of fluid collected rather than the time, i.e., the product Qt. If the length of the tube be doubled, the first dye will not appear until twice the volume of water has passed through the system. Thus the basic variable is the fraction of bed volume displaced, i.e., $Qt/V = \tau$. Making these substitutions in equation 1, we obtain the universal relation that the fraction of dye in a given sample for $\tau > 0.5$ is

$$F(\tau)d\tau = C(t)\ Qdt/l = d\tau/2\tau^2$$

(Equation 2)

as before for $\tau < 0.5\ F(\tau) = 0$. This is a universal expression which applies to all uniform bore circular cylindrical tubes irrespective of their dimensions provided the flow is streamline.

It seems scarcely likely that any vestige of organized flow of the type just discussed will remain in the circulation shortly after injection. Even if the settling out effect, and the nonuniformity of the injection process could be controlled, many factors will immediately act to randomize the indicator. A few bifurcations will be sufficient to destroy any regularity which might exist. Turbulence will occur at least in the heart. Most important, however, is the separation of the injected material in the capillaries and the later recombination in a completely un-

determined manner. For this reason, we must reject the purely deterministic point of view and turn to the methods of probability in our investigation of the most likely form of $F(\tau)$. We thus consider the nonrandom components in a nearly random situation.

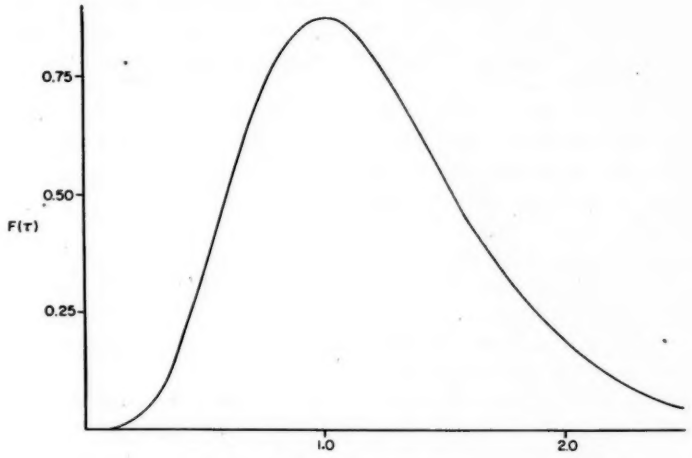

Fig. 2. Theoretical outflow of indicator for a serial system of 5 equal chambers in which uniform mixing is maintained. The abscissa is the fraction of the system volume displaced as fluid passes through the system. The ordinate is the fraction of injected indicator per unit τ interval appearing at a given τ instant.

The so-called "stochastic" approach based on probability considerations is to consider the type of $F(\tau)$ which would occur if the dye elements were being carried through a series of uniformly mixed compartments. There seems to be little anatomical reason to adopt this point of view. Perhaps the outflow from the heart might approximate the situation of two chambers, but nevertheless the advantage of this so-called "washout" model is a theoretical one. From the statistical point of view the dye particles progress through the chain of compartments in a series of jumps. In each compartment a particle makes many trials before it can escape because of the uniform mixing in the compartment. When it finally escapes, this is a rare event. The probability is proportional to the ratio of the flow rate through the compartment divided by its volume. The process of escape from any compartment is not controlled in any way by what happened in previous compartments. In general, when it leaves the n'th compartment, there have been n successes. This problem is a special case of a so-called "Markov chain."[2] It is completely analogous to the problem of radioactive decay of a nonbranching chain of nuclides in equilibrium and its solution is given in standard texts on radioactivity. If all of the dye is initially uniformly mixed in compartment one, then for n compartments

$$F(\tau)d\tau = \sum_{i=1}^{n} f_n \frac{f_i^{n-2}}{f_i \prod_{\substack{j=1 \\ j \neq i}}^{n} (f_i - f_j)} e^{-\tau/f_i} d\tau$$

(Equation 3)

where f_i is the fraction of the system volume in compartment i.* If the compartments are all alike the solution becomes

$$F(\tau)d\tau = \frac{n(n\tau)^{(n-1)}}{(n-1)!} e^{-n\tau} d\tau$$

(Equation 4)

Since for n compartments we are dealing with the probability of n-1 successes in a series of in-

*The π in the denominator signifies "the product of a set of factors each corresponding to a given integral value of j ranging from 1 to n but omitting the factor in which j and i are alike."

dependent equally likely trials of a rare event (i.e., the successive escapes from all but the last), the fact that we have obtained the Poisson law of probability is not surprising. In general, we should consider the Function $F(\tau)$ as a typical statistical frequency distribution function which, when multiplied by $d\tau$, gives the fraction of the total dye elements appearing in a given small class interval $d\tau$ of the independent variable τ.

Figure 2 shows a typical plot of $F(\tau)$ for a system of five equal compartments. It is evident that the curve has some resemblance to typical curves of the outflow of dye from the pulmonary circuit in man and in the dog. This led Newmar and his associates to postulate that often dispersion curves were adequately described by the "washout" theory.[10] It seems, however, that this agreement is the result of a statistical accident. One can scarcely believe that the movement of dye through a capillary bed and extensive system of branching vessels is the result of a small number of successes in a series of trials of a rare event. Instead of a few large jumps and long pauses in the progressive movement of the dye elements, it is eminently probable that a closer representation is given by a theory that considers a very large number of rapidly occurring small jumps. Under these conditions, the probability theory predicts that the particles will execute a "random walk."[2] Random walk problems have received considerable attention in the literature, but most mathematicians consider the situation of a fixed probability distribution for the jumps. This classical version of the random walk will serve as a first approximation to the physiological situation. We will consider it first and discuss the corrections to the theory separately.

A good experimental illustration can be obtained by filling a long uniform tube with 3 mm. glass beads, allowing water to flow through it and injecting a small amount of dye in one end. At the beginning the dye forms a small compact slug at one end of the tube. As the fluid moves through the beads some dye elements begin to move ahead and others to lag behind. The distribution of dye at various distances along the tube appears to be Gaussian, i.e., that of the normal curve of error. As the center of gravity of the distribution proceeds along the tube with essentially the same velocity as the average fluid velocity the dye spreads out and the standard deviation incerases in proportion to the amount of fluid which has passed through the tube. At the end of the tube the beads suddenly come to an end and so the dye can only cross the boundary once, being immediately swept away. A dye element is thus said to execute a random walk terminated by a first traversal. Because of the effect of the end of the bead column and because the standard deviation increases with the volume of fluid which has passed, the distribution of dye among the various fractions collected will not be Gaussian. The relation which can be derived from mathematical considerations[2] is

$$F(\tau)d\tau = \frac{e^{-\frac{(1-\tau)^2}{\varkappa^2\tau}}}{\varkappa\sqrt{\pi}\,\tau^{3/2}}\,d\tau$$

(Equation 5)

The randomizing constant $\varkappa$ represents $\sqrt{2}$ times the standard deviation relative to the mean of the dye distribution which would be obtained if, after the displacement of one bed volume, the experiment were stopped and if the various fractions could be removed from the tube and their label content determined without disturbing the distribution. $\varkappa$ is expressed in units of τ. In deriving this relation it is assumed that the center of mass of the distribution moves with the same velocity as the liquid, i.e., the probability distribution for the movement of the dye elements relative to the fluid is the same forward as backward.

The classical random walk problem is of general interest. In the theory of games of chance it is often called the Gambler's Ruin problem, since it describes the progress of a game in which a player contends with an infinitely wealthy opponent who has the odds in his favor. It has been used in physics to describe the Brownian movement of a particle. The background mathematics is found in a number of standard texts.[2] The problem is a special case of a Markov chain but is often developed as a limiting case of the random movement of particles between isolated points on a line as the points become infinitely numerous and close together. Particles are permitted to jump to a distant point or to neighboring points. It is required that no jump is in any way affected by previous jumps. Jumps of different amounts are not required to be equally probable but whatever the pattern of probability of the jumps in the classical version of the problem it must be the

same for all. The jumps are random in that their magnitudes cannot be predicted but merely the relative likelihood of a given sized jump. Jumps forward can be more frequent or less frequent than jumps backward. The degree of asymmetry will govern the rate at which the centroid of the population will move. The rate of increase of the standard deviation will be governed by the root mean square of the magnitudes of the jumps and the rate at which they occur. This will be determined by the magnitude of the proportionality constant in the denominator of the exponent. This will be the randomizing constant $\varkappa$ in equation 5. In a fine grained bed $\varkappa$ would be larger than in a coarse grained one, and ideally determining $\varkappa$ would yield valuable information.

Comparison of theory and experiment (Fig. 3) indicates satisfactory agreement in the case of a long thin tube filled with glass beads, but this is quite unrealistic as a description of the flow through a physiological labyrinth such as the pulmonary circuit. In the first place, the shape of the bed plays a role. The outflow of dye from a spherical vessel is shown in Figure 4. The water entered a spherical bulb flowed through the same size beads as in the tube experiment and was col-

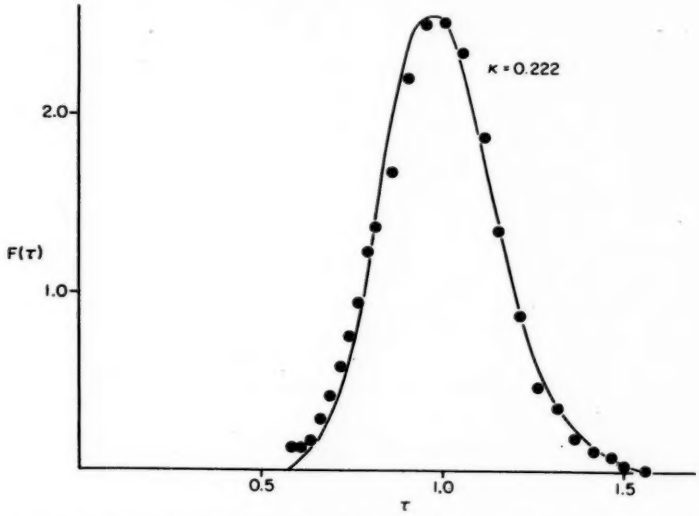

Fig. 3. Comparison of experimental results for outflow from a long cylindrical bed of uniform glass beads (●) with a theoretical one dimensional random walk curve (—). The water volume in the bed was 193 cc. See Figure 2 for ordinates and abscissas.

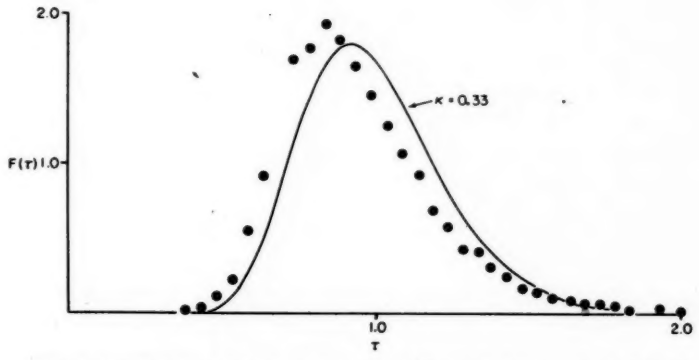

Fig. 4. Similar comparison (see previous figures) for a spherical flask containing 3 mm. beads. The water volume was 193 cc.

lected immediately at the outflow. The result systematically deviates from the random walk hypothesis, but it serves as a first approximation even though in this case the dye elements are not confined into a linear path for their excursions. The value of κ is significantly greater than for the linear tube case although the same size beads were used. Some improvements of fit could probably be achieved by introducing an effective bed volume and starting time, adopting the variable τ' which would be the fraction of the *effective* bed volume which has flowed through the system.

When a deliberate attempt was made to produce a severe distortion of flow from that of a straight tube the shape of the curve could be strongly influenced. Instead of allowing the fluid to emerge directly from the bulb, a tube was inserted so that its orifice was at the bulb center. Under these conditions (Fig. 5), there was a large increase in the value of the randomizing constant. Furthermore, to fit the theoretical curve, the volume of the water in the bulb could no longer be used but instead an effective volume 12 per cent greater had to be introduced, together with an effective starting time at $\tau = 0.1$.

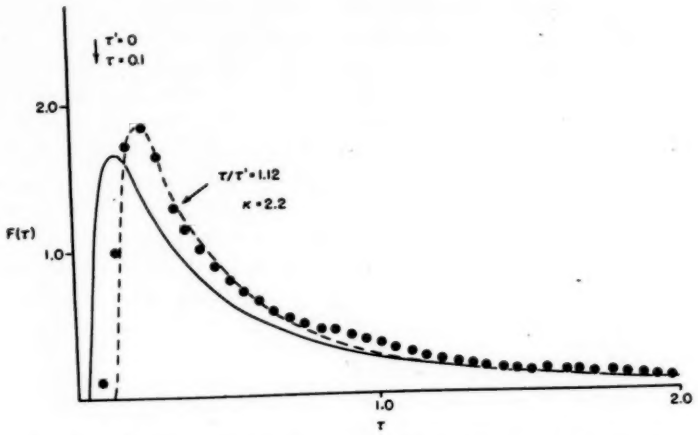

Fig. 5. Spherical flask (Fig. 4) with exit pipe projecting into the center.

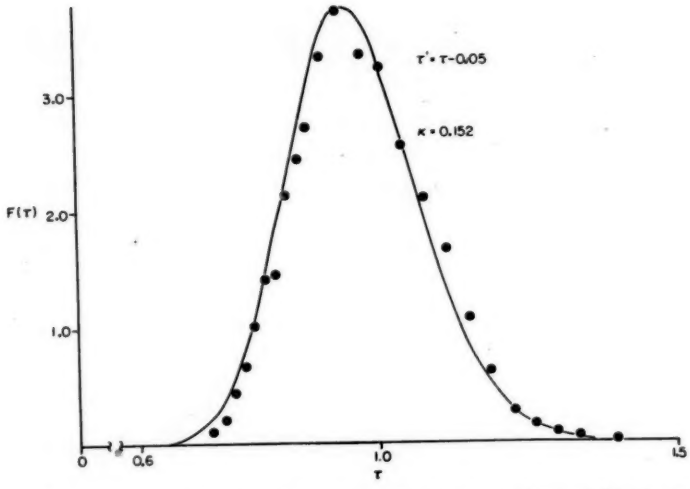

Fig. 6. Same as Figure 3, but dye injected in the rubber supply tube 71 cc. upstream.

The preceding experiments all involved a uniform randomizing structure in the bed. Dye was injected directly at the inflow, traversed a collection of uniform beads, and was collected with as short an outflow tube as possible. This, of course, ignores the fact that in a physiological system the volume of the vessels leading into the system is appreciable. Often, also the material must be collected through a long catheter, whose dispersing effect on the dye must be considered. Since the sum of the randomizing effects of a series of different dispersing systems should be independent of the order in which they occur, a long inflow or a long outflow tube should produce similar effects. To investigate the effect of a long entrance tube, the cylindrical container containing the 3 mm. beads was equipped with a 3-meter rubber tube of approximately 5 mm. I.D. Dye was injected into the upstream end and the outflow curve determined (Fig. 6). There is also a small 5 per cent correction for effective injection time. The curve is a fairly good fit to a random walk relation using a value of $\varkappa$ derived on the assumption that the equivalent $\varkappa$ for the tube is negligibly small. Since the flow through the tube is not well approximated by a random-walk relation, a good deal of further research must be done before such an effect can be entirely understood.

It was recently suggested by L. J. Savage and the author[14] that the one-dimensional random walk might serve as a first approximation to the problem of capillary outflow. Applications of the theory to physiological data are limited as yet because most of the curves have been taken from beds of unknown volume, so that at least two constants can be invoked in fitting the data. This gives more latitude than the theorist deserves. Figure 7 shows a comparison between the theory and experiment for data obtained by Hamilton et al.[5] who perfused an isolated heart lung preparation in the dog. A rather satisfactory agreement was obtained using a value of $\varkappa = 0.5$. However, a rigorous test of fit is not possible because the readings were obtained by enlarging the small figure in their paper which produces some transcription error. A similar curve obtained more recently by Howard, Dow, and Hamilton[6] is closely similar if the appropriate scale adjustments are made. Again a fair agreement is obtained. A second experiment which they performed on the same preparation gave essentially the same result. Unfortunately, a comparison between the effective bed volume and the true volume of blood in the pulmonary circuit is lacking since the latter volume was not obtained. Analysis of the curves using the flow rates, which they actually determined, yields only the effective volume.

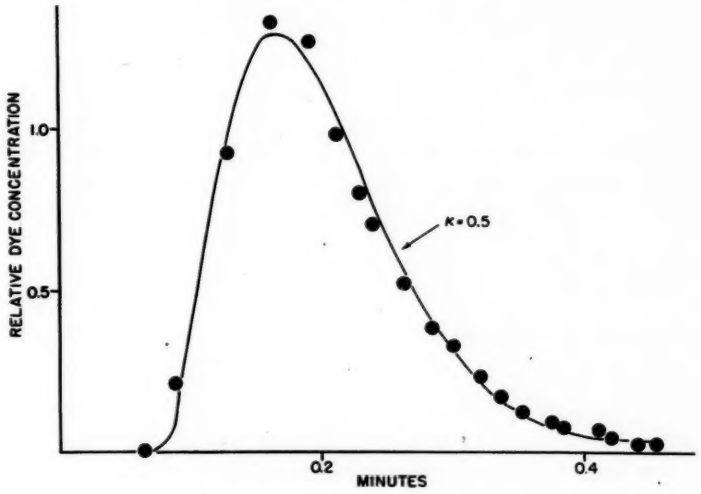

Fig. 7. Data of Hamilton *et al*, for perfused isolated heart-lung preparation in a dog.

When we turn to curves obtained on the lungs alone a different situation is observed. Three in-

jections in one preparation and two in another gave similar results if the probable difference in the cardiac output and volume of pulmonary blood in the two dogs and the variation in amount of fused an isolated canine liver preparation. The data were obtained in the course of other recently published studies on colloidal gold removal by the liver[7] (Fig. 9). Although the volume of blood

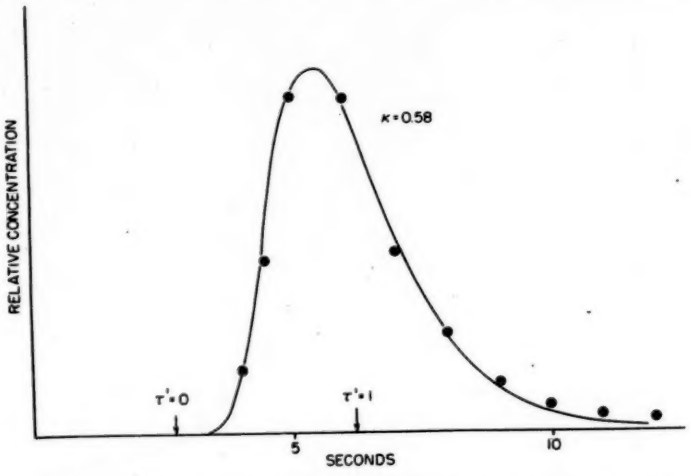

Fig. 8. Data of Howard, Dow, and Hamilton for perfused canine lung without the heart.

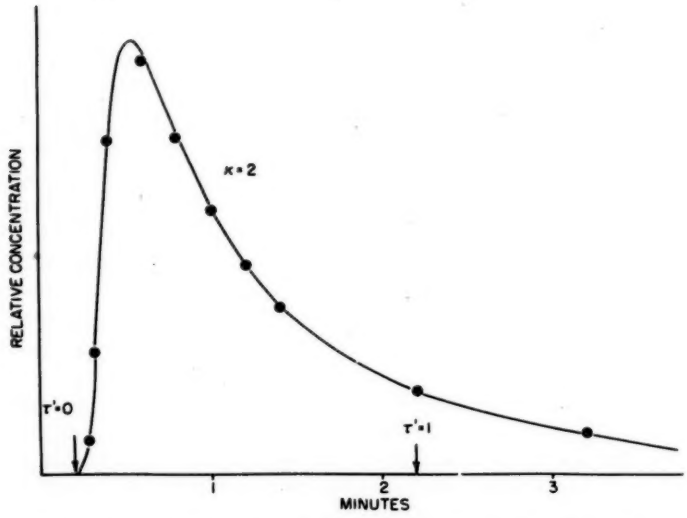

Fig. 9. Data of Little and Kelly for a perfused isolated canine liver.

injected dye are taken into account. A representative curve together with a fit to the random walk expression is shown in Figure 8. It is seen that to obtain a satisfactory agreement a considerably delayed effective injection time must be introduced. This is also true but to a somewhat lesser extent for a curve obtained by Little and Kelly who perin the liver was not known, the value of 240 cc. required to fit the data using their lowest flow rate seems high even for the approximately 16 Kg. dogs which they used. Because of these factors the one-dimensional random walk relation is probably only a first approximation to the true curves, and may often require the inclusion of effective

volumes and injection times in its use. A more complete analysis would consider the movement of dye in terms of a more general Markov problem, but first it would be necessary to get a better idea of the variation in probability distribution of the jumps of the elements of dye as they traverse the circulation.

In addition to the form of the outflow of dye during a single perfusion experiment, we must consider the manner in which recirculation is taken into account before we can discuss the actual process of circulatory mixing. Time will not permit the discussion of recirculation problems in general, but the familiar situation where indicator is injected essentially into the right heart, and samples are taken from the aorta or a close tributary will be sufficiently illustrative. The first step in the analysis is to relate events upstream at some point 1 to events downstream at 2. We will suppose that a small amount of dye is injected suddenly at 1. Let the average concentration of dye be $a(0)$ and the mean duration of the injection $\triangle t$. The product of these two factors will be the area under the injection curve. This area, when multiplied by the flow rate Q, will give the amount of indicator injected. We will consider only the case where the injection curve is a very high and very narrow spike. If the dye is injected at zero time, then in the limiting case where the height is infinite and the width infinitesimal (the case of the 'so-called "delta function"), the time distribution of dye downstream will be the distribution of traversal times, i.e., $F(t)$ and so the concentration at any time t is given by the relation

$$a_2(t) = a(0)\,\Delta t F(t)$$

(Equation 6)

This will relate events at 2 to those at 1 provided that there is no dye between 1 and 2 at the moment of injection. If we consider a more general problem, suppose that the time relation upstream is not that of a single spike but a continuous variation given by the relation $a_1(t)$. Then downstream at 2 we will have

$$a_2(t) = \int_0^t a_1(t-T)\,F(T)\,dT + \text{effect of dye}$$

(Equation 7)

between 1 and 2 at or before $T = 0$. The variable of integration T represents the various time delays of the individual paths between 1 and 2. The second term will be zero if there is no dye in the system before the experiment is begun. The integral is known as a "convolution integral." It is of general utility when it is desired to consider at some point downstream what the effect will be of the smearing of the earlier time relations occurring at some point upstream, when there are the variable traversal times between the two points.

If recirculation is complete, it is postulated that points 1 and 2 coincide in a closed circuit, becoming a single observation point so that

$$a(t) = \int_0^t a(t-T)\,F(T)\,dT + \text{effect of first circulation.}$$

(Equation 8)

The second term is required because all recirculation effects at the point of observation arise from effects one circulation earlier. The dye already in the loop during the previous circulation will be included only after one initial circulation has occurred. The added term is merely the time relation for one traversal of the labyrinth which for a sudden injection is given by equation 6 so

$$a(t) = \int_0^t a(t-T)\,F(T)\,dT + a(0)\,\Delta t\,F(t).$$

(Equation 9)

This is an integral equation in which given $F(t)$ and $a(0)\triangle t$ we find $a(t)$. It is so called because the unknown function to be found in solving the equation is related to its own integral which includes the function $F(t)$ called the "kernel."

The solution of the equation involves a principle which is of such potential utility to circulatory physiology that some discussion is warranted. The considerations are based on the so-called "method of Laplace transforms" which is discussed in books on electrical transient analysis.[4] We multiply both sides of equation 9 by e^{-kt} and integrate with respect to t from 0 to ∞. In so doing we convert a (t) to its Laplace transform $\alpha(k)$. Since we have introduced the variable k and removed t by integrating it out, the transform is a function of k. The reason for this maneuver is that the transform of a convolution becomes the product of the transforms of its components

which greatly simplifies the solution of the equation. Thus in equation 9 we have simply

$$a(k) = a(k)\,\Phi(k) + a(0)\,\Delta t\,\Phi(k)^{t}$$

(Equation 10)

where $\Phi(k)$ is the transform of $F(t)$. Simple algebra yields the transform of the solution of the equation, namely

$$a(k) = a(0)\,\Delta t\,\Phi(k) / [1 - \Phi(k)]$$

(Equation 11)

The transform is converted back to a(t) by the process of inversion, which is facilitated by the use of tables of transforms and their inverses which are available.[4,9] The first discussion of the method of Laplace transforms in the study of indicator dilution techniques was given by Stephenson.[15]

In the present case the quantitative discussion of complete circulatory mixing would require a knowledge of the kernel $F(t)$ for the entire circulation from the right heart through the pulmonary and systemic circuits and returning through the vena cava. This is not known but as a first approximation we could assume it to be similar to a random walk curve, taking for k the same value as for the pulmonary circuit. The result could then be reduced to time variables by the use of standard cardiac output and plasma or blood volume figures.

Although the random walk curve is based on a rational set of theoretical assumptions, the curves are not very different for a set of equal uniformly mixed serial compartments, and the transforms for these latter expressions are much simpler and easier to handle. We accept the form of equation 4, but consider n now rather than $\varkappa$ as an empirical parameter and choose τ rather than t for the independent variable, thus we have

$$F(\tau) = n(n\tau)^{(n-1)}\, e^{-n\tau} / (n-1)!$$

$$\Phi(k) = n^{n} / (k+n)^{n}$$

(Equation 12)

whose transform by simple algebra is

$$a(k) = a(0)\Delta\tau / [(k/n+1)^{n} - 1]$$

(Equation 13)

This can be inverted by separation into partial fractions yielding a (t). The case of $n = 4$ will serve as an illustration, thus

$$a(k) = a(0)\,\Delta\tau / [(k/4+1)^{4} - 1]$$

$$= \frac{a(0)\Delta\tau}{2} \left\{ 1 / [(k/4+1)^{2} - 1] - 1 / [(k/4+1)^{2} + 1] \right\}$$

(Equation 14)

From tables[4] this yields

$$F(\tau) = \frac{a(\tau)}{a(0)\Delta\tau} = 2e^{-4\tau} [\sinh 4\tau - \sin 4\tau]$$

(Equation 15)

Figure 10 shows a plot of this relation. A similar plot is shown for $n = 8$ (Fig. 11).

The theory describes recirculation phenomena at the right atrium but the majority of observations are taken in the aorta, and thus it is necessary to add the further smearing or dispersing effect of the pulmonary circuit. This is simply done by multiplying the transform in equation 14 by the additional transform for the pulmonary circuit. Again we assume the same form for the local portion as for the over-all circulation, but consider for the moment that the volume of the pulmonary portion is some fraction f of the total. The resulting composite transform for observations in the aorta is

$$\frac{a_a(k)}{a(0)\Delta\tau} = \left\{ [(k/n+1)^{n} - 1]\; [fk/n+1]^{n} \right\}^{-1}$$

(Equation 16)

For $f = \frac{1}{3}$ and for $n = 4$ or $n = 8$, the inverses are shown in Figures 10 and 11. As before, the solutions include only the component after one passage and the first passage contribution must be added. The entire solution is also included in the figures and the curves are not unlike the experimental results so often observed.

Many points remain to be investigated. Time will permit only a brief mention of a few. Little has been said about the transition effects which occur when two randomizing beds with widely different $\varkappa$'s are placed in series. What in general is the effective $\varkappa$ of the combination? The effect of two random walks in parallel is the same as that of a single equivalent one, only if the flow

is shared between the two members in proportion to their volume. What kind of linear random walk do we have if $\varkappa$ varies smoothly through the labyrinth according to some prescribed functional relationship? What can we say about the 3-dimensional random walk when the particle can wander sideways into regions of faster or slower flow?

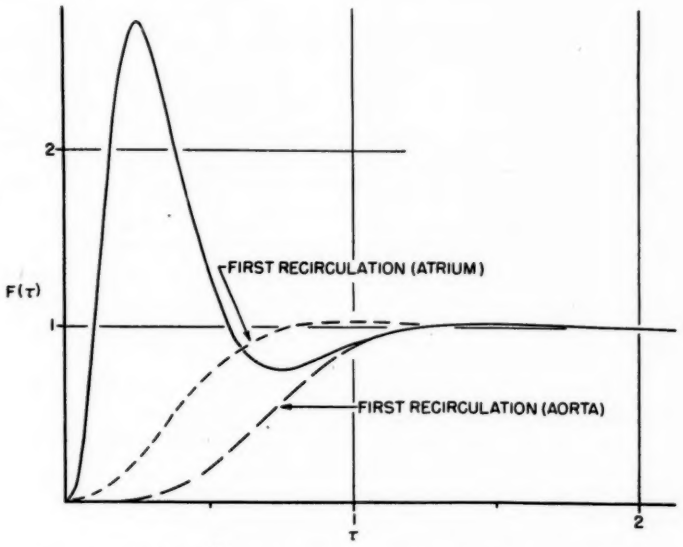

Fig. 10. Solution of the recirculation problem (see text). Solid curve represents theoretical dye curve for injection in the right atrium and observation in the aorta. Dotted curves represent recirculation observed in the atrium and aorta. Distribution in traversal times through the circulation approximated by the relation for the Poisson probability of three successes.

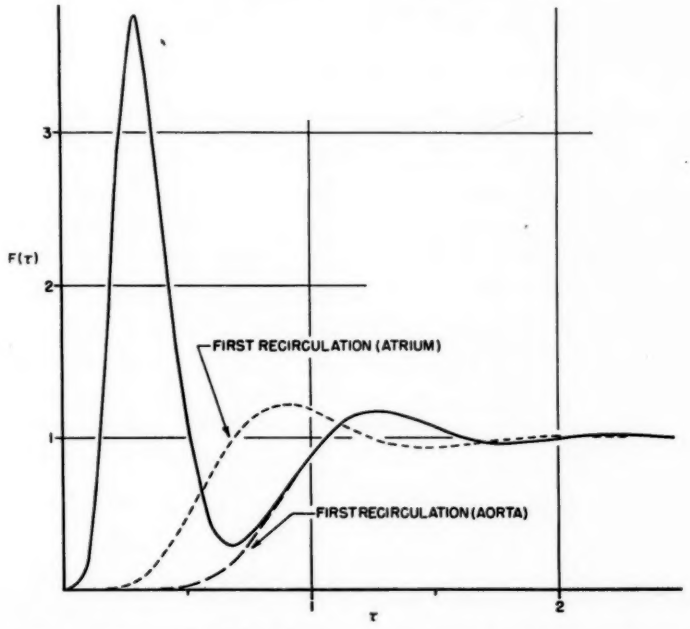

Fig. 11. Same solution as Figure 10 but for seven successes. Note the effect of the increased peaking of the traversal time distribution.

From these considerations alone we see that the present calculations are far from being a complete analysis of circulatory mixing processes. However, they seem to be at least partially successful, and it may be that a rational approach to the study of circulatory transients can be achieved in this way. In any event the results are interesting and the initial successes are sufficient to justify further work of this nature.

In conclusion, I wish to acknowledge the kind assistance of Drs. Howard, Dow, and Hamilton for the canine pulmonary outflow data, and Dr. Robert C. Little for the data on the canine liver perfusion. Some of the laborious numerical calculations were performed by Mr. George Atta of the ORNL Mathematics Panel.

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THE RELATION BETWEEN THE STROKE VOLUME AND THE PULSE PRESSURE

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IT would be well nigh impossible to trace the origin of the notion that the pulse pressure is directly related to the stroke volume. Every physician who places his fingers over the radial artery, does so with the firm conviction that the strength of the beat he feels is set by the amount of cardiac discharge. Our province in this symposium is to see to what extent this subjective impression can be set into quantitative terms. Near the turn of the century, the first step in such a quantitation came with the technology with which pressure excursions in man could be approximately determined in millimeters of mercury. Shortly after, Erlanger and Hooker[2] noted that when the pulse pressure, so recorded, was multiplied by the heart rate, the product tended to be reasonably constant. In those conditions where the cardiac output was presumed to be increased, the product was elevated. Conversely, when the total flow should have been low, as after hemorrhage, the product was decreased. This implied that the pulse pressure was varying directly with the stroke volume. Since cardiac output of man was not, at that time, measurable, physiological conclusions reached on the basis of the change in product were entirely speculative. The quantitative testing fell to workers using the most common method of assessing stroke volume, the cardiometer. On the basis of their work on open-chest animals, they rather promptly discarded, almost *in toto*, the notion that stroke volume could be predicted with any reasonable accuracy from the pressure change. Explanations for this seemingly illogical failure were not clearly put, which perhaps explains why newer generations of physiologists have not remained convinced that the problem is hopeless.

Upon hindsight, we could construct a rather forceful story for the cardiometer workers, with most of the points used, taken from work already published before their investigations were done. In 1880, for example, Roy[12] reported volume-pressure measurements for segments of aortas which showed that the volume equivalent of a given pressure change was different at different pressure levels. This nonlinearity of the volume pressure curve should have been obvious. Even if the aortic wall followed Hook's Law in its stretch, and showed a linear tension-length relation, conversion of tension to pressure would be through the first power of the radius, and of length to volume through the square of the radius, so that the volume-pressure relation must now be curved. Since the aorta does not follow Hook's Law, the curvature is even more pronounced. The volume gain per unit pressure rises therefore increases as pressure rises from 0 to 30 to 40 mm. Hg, after which it becomes approximately constant. At a pressure level of about 100 mm. Hg, the volume gain becomes progressively less. This second inflection is attributable to the complex architecture of the aortic wall. The physician is certainly well aware of it, for he would not think of ascribing an enormously large cardiac discharge to a hypertensive pressure excursion of, say 270/130. For some curious reason, some experimental workers tend to ignore it, and assume a straight-line relationship between volume and pressure obtained at all working pressure levels.

Unfortunately, these inflection points do not lie at the same pressure levels in all individuals, so that there can be no common prototype curve applicable to all aortas. In addition, intra-individual differences in aortic size are quite large. In a group of nine dogs, for example, at a diastolic pressure of 75 mm. Hg, the total aortic volumes varied from 19 to 39 cc., the larger value representing a departure of 39 per cent from the mean of 28 cc. One factor contributing to this variation must be body size. We cannot tell from our data whether this variation with size would be better related to body weight or to surface area. Since we are accustomed to expressing cardiac output in terms of surface area, all arterial bed volumes were corrected to figures per sq. M. body surface. When the above dog aortic capacities were so

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revised, the largest error in the group was reduced from 39 to 29 per cent. Variability still remaining after such a size correction has been made, can be laid to congenital differences or to the effect of

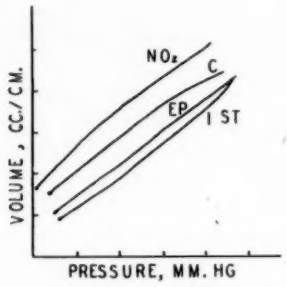

Fig. 1. Volume-pressure relations for a ring of ascending aorta as first removed from the body (1st), after several stretchings (C), after addition of epinephrine (EP), and after addition of $NaNO_2$ (NO_2).

age. Roy had mentioned that an aortic segment from a young individual showed a different distensibility pattern from that of an older person. This effect of age was later emphasized in greater detail by Hallock and Benson,[3] who, by publishing only average curves, gave the impression that the change was a progressive and orderly one. When we surveyed a group of forty-eight human aortas,[11] we found that the capacity change with age was only a trend, and that the degree of overlap between age groups was very large. This variation can perhaps best be stated by the fact that we found a "young" aorta in a man of sixty-eight, and an "old" one in a girl of eighteen. Roy reported something which I do not believe has been repeated since, that after a long wasting disease, the dog aorta showed the stretch curve of an "old" aorta. It is quite possible that the variations with age are reflecting not the actual years of life, but rather the history of the number of periods of disease and circulatory stress. Certainly the enlargement often attendant upon hypertension can be seen in both the young and old individual.

In this "aging" process, as the aorta increases its capacity, the relative volume gain per unit pressure rise, as related to this initial capacity, is diminished. This change, the physicist would clearly term a decreased distensibility. Yet when the actual volume-pressure relations for all forty-eight aortas were plotted on the same figure, the most constant feature between them was the actual volume gain, without any reference to initial size. This relative constancy is fortuitous, for it is this actual volume increase that we wish to know when we would quantitate the pulse pressure in terms of volume. What a relatively stable actual volume increase, in face of the marked differences in relative distensibility, would mean to the physicist can hardly be treated in the time allotted. Individual variation away from the average net uptake figures averaged 25 to 30 per cent for the human series and 15 to 20 per cent for the dogs. Intrinsically, then, if we use these average values to predict actual volume uptakes from the pressure change in living individuals, we can hardly expect an accuracy greater than this. Actually, the error has often turned out to be less, as though the autopsy series contained an undue number of "atypical" aortas.

The cardiometer workers were not protesting the use of the pulse pressure as a measure of stroke volume between individuals, however. Where they had failed was in finding corresponding changes in pulse pressure when the stroke volume was acutely changed in the same dog. This implies that arterial distensibility shows physiological variation. The arteries have muscular walls. Even the aorta has variable amounts of smooth muscle. There is no reason to suppose that this muscle will not be influenced by nervous and humoral stimuli. MacWilliam, in 1902,[6] reported that when the aorta was first removed from the body, it was small and quite firm. After manipulation, it became larger and flabby. Stretch curves made on aortic segments in the two states showed that the firm aorta had a greater relative distensibility. The sort of changes he was observing can be illustrated by some recently recorded volume-pressure relations for a ring of dog ascending aorta, as shown in Figure 1. Upon first removal from the body (1st) the ring was small. After several stretchings, it was considerably larger. After the addition of epinephrine to the bathing medium, the ring again became contracted, while after $NaNO_2$ was added, it was relaxed. Just as was the case of the capacity changes in aging, however, the actual volume-pressure curves show a fairly good parallelism despite these tone changes. This means that an average volume uptake table can discount the effect of tone changes.

When Dr. Hamilton and I started on this problem, it seemed that the really major source of error had not even been considered. So far I have been speaking as though the only variable between pulses was the height of the pressure rise. Actually, in different circulatory conditions, the central pulse contour of the dog may take quite divergent forms, with the peak falling in early, mid or late systole, and the duration of ejection varying from 80 to 230 millisecond. The pulse pressure itself cannot be expected to signify these different contours. The volume uptake of the arterial bed should instead be related to the time-pressure area under the systolic portion of the pulse curve. But the problem is even more complex. The heart is ejecting into one end of a long network of tubes, all of which comprise the arterial receiving chamber. At the very first of systole, only the ascending aorta is accepting blood, and a given volume input must produce an inordinately large pressure rise. As the pulse wave moves out the vascular tree, the size of the reservoir is constantly increasing. Some means must be found to weight the parts of this time-pressure area for the progressive increase in reservoir size.

To evaluate the dimensions of the receiving chamber, we need the net volume increases per unit pressure rise, and the various pulse wave transmission times at different pressures, for the arterial beds. The former were assembled from evidence based on pressure rises following injections of known amounts of blood, or on tension-length curves obtained by stretching rings of vessels.[10] For the latter, transmission times were recorded to various arteries easily entered by manometer needles, and the values for other beds assembled by reasonable extrapolations. Next, all vessels lying the same time-distance from the heart were grouped together, to be treated as though they comprised a single tube. The different values for the volume uptakes of these vessels were also lumped by the same pattern. Hence we can know, for this single tube, the volume gain per unit pressure rise for each millisecond of its length. Now if we assume that a given central pressure pulse will pass through this theoretical tube without any change in form, we can calculate, moment by moment, the volume uptake of all parts of the tube, which can be summated for the total volume uptake.

This total uptake does not represent the stroke volume. As the pulse wave reaches the terminal arterioles, drainage is accelerated because of the increased pressure. The extra blood so lost must come from the reservoir, and rob it of some of its uptake. This drainage is estimated in a rather gross fashion. We assume that the outflow rate will be linearly proportional to pressure, less 20 mm. Hg, which is taken as the level at which flow ceases. This pressure level presumably is altered under different physiological conditions, but there are no convenient handles by which it can be corrected to meet the specific circumstance. Let us also say that all of the above determined uptake drains from the arterial reservoir during the succeeding diastole. Systolic drainage then can be calculated by the ratio of the time pressure areas under the systolic and diastolic parts of the pulse, multiplied by the uptake. Yet not all of systole is available for systolic drainage, for time is required for the pulse wave to reach the terminal arterioles. Transmission times to the various large drainage beds were obtained by extrapolations. Next, the fraction of the total cardiac output that normally flows through each bed was arbitrarily assigned, using values taken from the literature. The products of the flow fraction and the transmission time for all beds were summed, and averaged, to give a flow-weighted time value that we call Tw. This Tw then represents the mean time required for the pulse wave to reach an arteriole that carries an average amount of the total flow. This term Tw has a specific definition. The use of the symbol for times values differently derived can seemingly only lead to confusion.[13]

The effective systolic time for drainage can now be taken as the total systolic duration less Tw. Having calculated the total volume of systolic drainage, a fractionation between beds can be made, and the time course of systolic drainage determined. When the uptake and the drainage curves are summed, we should have the total ejection curve.

When we desire only the stroke volume rather than the ejection curve, the calculation need not be so elaborate. The whole stroke volume must be delivered at the time of the incisura, when the valves close. We should know the uptake values for the different regions of the composite tube at this moment. For simplicity, the tube is treated as composed of four segments only, each segment beginning its uptake when the pulse wave has reached the mid-point.[4] These half-times for

each segment were tabulated. The pressure height for each segment, which should be proportional to end-systolic uptake, is obtained by laying back this time from the incisura on an actual pulse. The segment uptake for this pressure height can then be obtained from a table. When this process is repeated for each segment, we will have four uptake values which can be added for the total uptake.

The whole technique rests on the accuracy of transmission times, and the applicability of our set of average time values to different animals, or to different physiological conditions in the same animal. When we studied the arch-iliac transmission time for a number of animals at a given pressure level, we found the individual variations to be about 10 per cent of the mean. We were not able to establish any systematic deviation according to body size, indicating that such a trend was being masked by a larger variation attributable to tone changes and the other physiological factors which influence the wave velocity.[5] We therefore have no data to allow a correction for body size, nor can we safely speculate as to how these times would be affected when both wall thickness and vessel length are increased as the body becomes larger. As to the physiological variations, we have seen that when tone changes occur, they affect the relative distensibility, which conditions the pulse wave velocity much more seriously than the volume uptake values. It would seem safer to apply a standard set of transmission values than to make corrections every time the pulse wave velocity might change in a particular artery.

Alexander[1] did find a trend of arch-femoral transmission times according to body weight. When he calculated stroke volume with time values revised for each animal, the average difference between the values obtained and those given by our single table was some 6 per cent. The difference proved to be random rather than systematic. He concluded that the use of average time values had not introduced a really serious error, a belief we had had from the beginning.

We hear much of a lengthening, with the development of tortuosity, in the vessels in older humans. In our eighty-three aortas, length changes were certainly less clear than were changes in diameter, and individual variations not nearly so large. We should remember, also, that when age and/or hypertension produces such a lengthening, it also produces a thinning of the wall and a decreased relative distensibility. The resultant increased pulse wave velocity might easily compensate for the greater length.

The quantitative justification of the volume calculations, as outlined, requires a check with a recognized measure of cardiac output, done simultaneously. No other procedure, however, measures individual stroke volume, but rather gives a value for mean flow over a relatively long period of time. Since dog pulses may vary quite appreciably from beat to beat, the selection of single pulses which shall typify the whole time span is a problematical procedure. Making as reasonable guesses as possible as to pulse selection, the contour calculation has been compared with the Fick or the dye injection techniques in four different laboratories, and the results in three are fairly similar. The average disparity between the two procedures is from 10 to 15 per cent.[7] The results of some 400 such comparisons, done at Georgia, are shown in Figure 2. These points cover a wide range of experimental conditions, including both hypertension and hypotension, acute and chronic.

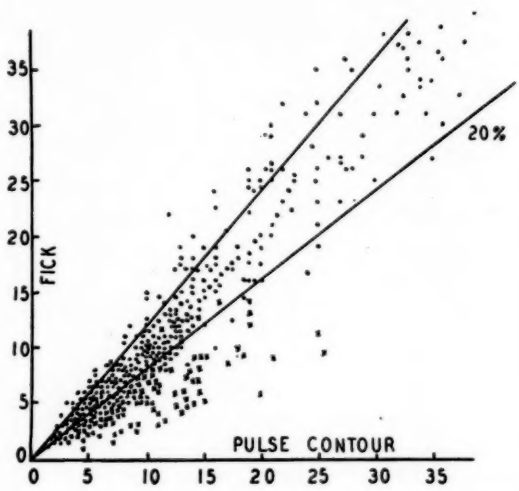

Fig. 2. A comparison of the stroke volumes obtained by the Fick procedure, using oxygen, and the Pulse Contonur calculations for 400 experimental cases in dogs. The lines represent a spread of ±20 per cent from the line of identity. For the difference between points and crosses, see text.

These points are fairly sharply divided into two clusters. The dots group around the line of identity without a systematic deviation. Seventy-three per cent of them lie within the limits of ± 20 per cent. These are from a great many different

circulatory states in dogs in relatively good condition. The crosses, on the other hand, show a large number of poor fits with the Fick results, with the disagreement always in favor of an excessive pulse pressure for the corresponding stroke volume. The aberrant pulse contours tend to have a common form, with a very sharp anacrotic pressure rise, poorly sustained pressure in later systole, and a relatively brief duration of ejection.[8] These pulses come, in the main, from animals in what we might term a "normatensive" shock state—animals with but slightly subnormal pressure levels, suffering from only a mild blood volume reduction, but with cardiac output levels that can only lead to death. It is curious that as an open-chest dog deteriorates, it is prone to enter this peculiar state. Why the distensibility behavior of the arterial bed is changed so radically, and by what mechanism, is a most intriguing problem.

The fact that the pulse contour calculation is not applicable to all situations is of course disappointing. But we must not let this disappointment blind us to the fact that if we discard pulses of the aberrant form, and avoid conditions where such aberrancies might appear, the technique can yield a great deal of most profitable information.

In developing a case for the cardiometer workers, we have presented a number of points that could have sufficed to strengthen their case. However, we have also attempted to discredit each of them. The only good reason remaining for the reported lack of correlation was in the assumption that the pulse pressure could typify a pulse contour. It is interesting in this regard that when the method was applied to some records published long ago by Yandell Henderson, the calculated stroke volumes agreed quite well with those he obtained with a cardiometer.[9] Yet even this assumption should have allowed a recognizable trend between stroke volume and pressure change in the earlier work, unless enough of their experimental animals had entered the shock state. In addition, I am often reminded that the favorite device for adjusting the pressure level in their preparations was an occlusion of the aorta, which of course radically altered the size of the receiving chamber. Curiously enough, an increased pressure excursion, with relatively the same stroke volume, produced by such an occlusion seems to have been one of the crowning pieces of evidence that a relation between the two could never be expected.

How much of this methodology is applicable to the human? When we derived the dog technique, central pulse contours in the human were not being recorded. We did collect a series of brachial pulses, recorded directly from the artery at the same time that a Fick determination of cardiac output was being done. A large portion of these were borrowed from the files of Dr. Andre Cournand. With not a great deal of confidence in the results, we empirically constructed a pulse contour method for these pulses. The lack of confidence stemmed from the fact that these pulses often showed only a superficial resemblance to what we might expect of a central pulse, if a human resembled a dog. From the records I have seen in Dr. Wood's laboratory at the Mayo Clinic, this premonition seems to have been well founded. These brachial pulses usually did not show any remnant of the incisura, or even any constant landmark which could be taken as marking the end of systole. The brachial pressure values themselves were heightened to variable degrees by fusion with reflected waves. Curiously enough, despite these handicaps, the contour calculations gave an average fit of ± 20 per cent from the Fick value.[11]

If variability in the basic data were comparable, central human pulse contours would seem better suited to accurate volume predictions than are dog pulses. Changes in contour seem far less extreme, heart rates are much more stable, beat-to-beat variations in pressure much smaller, and the length of systole much longer and more stable than for the dog. The long systole minimizes the effect of the expansion of the arterial reservoir during systole. On the other hand, it increases the systolic drainage fraction of the calculated stroke volume. We have followed with considerable interest the efforts of the Mayo group to construct a contour method for the central pulse of man.[13]

Limited as we were to brachial pulse records, we doubted that the accuracy achieved by application of the contour calculation methods to an unsuitable pulse, was rewarding enough to continue the approach. We therefore returned to the original Erlanger and Hooker notion that the brachial pulse pressure, *per se*, could be quantitatively related to the stroke volume. As we have said, this implies a stereotype contour form.

From the pulse pressures and the known stroke volumes, we derived a set of volume factors

TABLE I. FACTORS FOR THE PREDICTION OF STROKE VOLUME, PER SQ. M. BODY SURFACE, FROM THE PULSE PRESSURE

Pressure	Volume Factor	Pressure	Volume Factor	Pressure	Volume Factor
mm. Hg.	cc.	mm. Hg.	cc.	mm. Hg.	cc.
20	0	100	81	180	140
30	10	110	90	200	148
40	21	120	100	220	155
50	31	130	108	240	161
60	42	140	115	260	167
70	52	150	122	280	173
80	62	160	128	300	179
90	71	170	134		

(Table I), corrected for body size and for the inflections in the aortic distensibility curve. If, for example, the brachial pulse pressure was 120/80, then the predicted stroke index, from Table I, would be 100-62, or 38 cc. As a rule of thumb, over the usual pressure range, 1 mm. Hg, pressure rise is about equivalent to 1 cc. stroke volume/sq. M. body surface. The average error of these predictions was about 25 per cent. How good a fit this is depends upon one's viewpoint. If workers with the ballistocardiogram got such an agreement, they would be quite content. For the experimental investigator it leaves much to be desired.

For the diagnostician, a prediction with this error should still furnish a great deal of useful information as to the direction and general magnitude of cardiac output changes. After all, the flow changes with which he is most concerned tend to be larger than the intrinsic error. In case of critical, or small, changes in flow, corroborative support from a direct cardiac output determination would, of course, be necessary.

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DIAGNOSTIC PROCEDURES IN DETERMINING CONGENITAL CARDIAC DEFECTS

Aside from a thorough history and physical examination, the most important single diagnostic procedure which can be done in the physician's office is careful fluoroscopic examination. A good deal will be missed if the fluoroscopy does not include the right and left anterior oblique views in addition to the usual posterior-anterior view. Fluoroscopy will demonstrate enlargement of the individual cardiac chambers and will give information as to the contour and activity of the pulmonary vessels.

The electrocardiogram, especially with precordial leads, is useful in demonstrating ventricular hypertrophy. Right axis deviation is found in the tetralogy of Fallot, left axis deviation in tricuspid atresia.

Catheterization of the heart may be helpful in a difficult case. A special catheter is inserted into an arm or leg vein and gently advanced under fluoroscopic visualization to the right auricle. The catheter is then passed through the valve into the right ventricle and, usually, into the pulmonary artery.

In the case of an over-riding aorta, the catheter may be passed from the right ventricle directly into the aorta. It is sometimes possible to pass a catheter through an auricular or ventricular septal defect. Blood samples are taken from the different chambers and analyzed for oxygen content. Information may thus be obtained concerning left-to-right shunts, such as patent ductus, by analysis of the oxygen content of blood samples drawn from the right ventricle and pulmonary artery. (The latter sample will have a greater oxygen content because of the flow of oxygenated blood from the aorta.) The presence of auricular and ventricular septal defects will similarly be demonstrated. By a manometer attachment to the catheter a recording of pressures during systole and diastole is made. This is helpful in showing the presence of pulmonary stenosis when a recording of the pressures in the right ventricle is high and in the pulmonary artery is low.

Catheterization must be carried out by a well-trained team in a hospital, and usually general anesthesia is necessary.

Angiocardiography is a procedure in which a radiopaque dye is injected into a vein and with a special camera x-ray films of the heart are taken in rapid sequence. These films visualize the great vessels and individual chambers of the heart as they are filled by the dye.

Angiocardiography is particularly useful in the diagnosis of defects such as coarctation of the aorta. It may be of some help in the diagnosis of transposition, truncus arteriosus, tricuspid atresia and anomalous venous drainage. In an older, co-operative child, it may be carried out without anesthesia.

—From Diagnosis of Congenital Cardiac Defects in General Practice, American Heart Association

QUANTITATION OF STROKE VOLUME CHANGES IN MAN FROM THE CENTRAL PRESSURE PULSE

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IN the preceding paper Dr. John Remington has given us an historical background to the problem of estimation of left ventricular stroke volume from an arterial pressure pulse. He has also presented some of his own work in this field, and has pointed out some of the difficulties he encountered in trying to apply this method to the study of man. I should like to present briefly a somewhat different approach to the problem. The work I shall describe was carried out in conjunction with Dr. Earl Wood, and others in the physiology laboratory of the Mayo Clinic.[4]

The pressure wave generated in the aorta and large arteries by the periodic ejection of blood into this system by the left ventricle is a complex phenomenon. Necessarily, in any attempt at the present time to quantitate volume changes, from a recording of this pressure pulse, must be considered only a first approximation, and the attempt is justified only if the results obtained possess a useful degree of accuracy, and if the attempt suggests new and more precise means of approaching the problem.

Inasmuch as the pressure wave traverses the arterial system with a finite velocity, pressure in this system is a function of at least two variables, namely, time in the cardiac cycle (T), and distance down the arterial bed (X). Pressure can be recorded as a function of time at a fixed point in the system. If a plot of pressure as a function of distance at certain specified time, could be obtained, namely the onset of systole and the end of systole, the difference between the mean pressures over the length of the system at these two instants would be the pressure change corresponding to the volume change which Dr. Remington has named the systolic uptake—that is, the volume of the arterial bed at the end of systole minus its volume at the onset of systole.

In order to calculate (P) as a function of (X) from a record of (P) as a function of (T), one must know (X) as a function of (T). The first derivative of this relationship $\frac{(dx)}{(dt)}$ is the pulse wave velocity, and this has been found to be an increasing non-linear function of (X), and varies considerably from one individual to the next. However, it was found that changes in mean pulse wave velocity between two points in the arterial bed (for instance between the brachial and the radial artery) are proportional to changes in mean velocity between any other two sites (aortic arch to femoral artery for instance). Thus changes in pulse wave velocity between two recording sites in the arterial bed can serve as an index to the time of transmission of the pulse wave down the whole system (Dr. Remington's Tw).[2]

The length of that portion of the arterial bed that acts primarily as a passive elastic chamber is arbitrarily assigned a value of 60 cm. Then on the basis of an average pulse wave velocity in the resting supine subject of 6 meters/sec. in this segment, transmission time over the length of the system (Tw) would be 100 milliseconds at rest. A proportionality factor, between this and the measured transmission time at rest, between the two recording sites, is then calculated and used to estimate subsequent values for Tw in that individual. The accuracy of the resting, assumed value for Tw is not at all critical to value for stroke volume obtained by this method, since only changes in stroke volume are to be calculated.

In Figure 1 is shown a pressure-time record recorded through a small catheter, the tip of which lay in the aorta, and another, whose tip lay in the femoral artery. The integrated mean pressure between the limits of the onset of systole on the central pulse and a point Tw back from this point, serves as an index to the mean pressure down the aorta and large arteries at this instant. The difference between the mean pressures down the system at the onset, and the end of

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systole, is the pressure increment which we have called the mean distending pressure, and which corresponds to the volume change—systolic uptake.

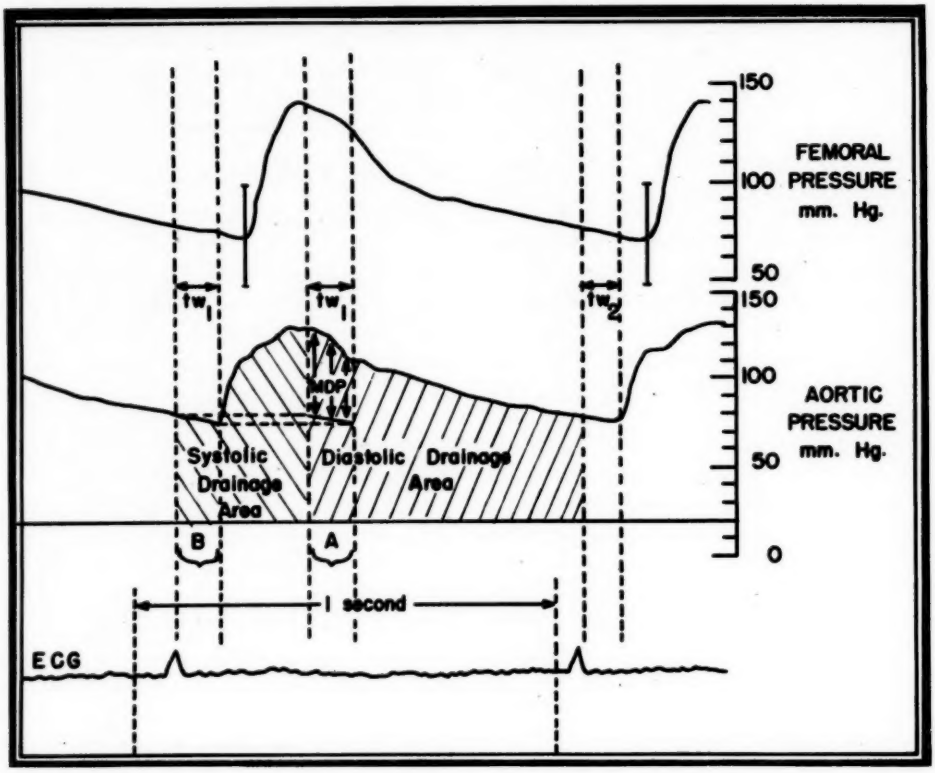

Fig. 1.

The other measurement to be made from the pressure record is also shown in this figure, namely, the ratio of the systolic to the diastolic time-pressure integral above 20 mm. Hg, corrected for the delay in transmission of the pressure wave to the periphery.

Dr. Remington has shown a plot of the volume-pressure relationships which they obtained by stretching rings of aorta from recently dead humans.[3] They found a marked individual variation in size and distensibility within any given age group. Although not evident in their plot of average values, examination of the individual values shows considerable variation in slope from one individual to the next. As a matter of fact, the data would indicate that the variation from linearity, when it occurs, is relatively small in comparison to the individual variation. Because of this, a linear relationship between volume and pressure is used as an approximation in the method being described.

In order to arrive at this proportionality factor relating change in pressure to change in volume for a given individual, cardiac output is measured with the subject at rest by an independent method (either the Fick or the dye method) while the pressure pulse is being recorded. Systolic and diastolic time-pressure integrals and mean distending pressure are measured, from each pulse during the independent cardiac output measurement, and averaged.

A sample calculation, that illustrates the application of these equations to the measurement of changes in stroke volume produced by exercise, will clarify the preceding paragraphs.

Person at rest:

Pmd = 39 mm. Hg (measured value)
Sa/Da = 0.48 (measured value)
SV (dye) = 127 cc. (measured value)

Equation 8:

$$SV = U + \frac{Sa}{Da}U$$

$$127 = U + 0.48U$$

$$U = \frac{127}{1.48} = 86 \text{ cc.}$$

Equation 4:

$$k = \frac{U}{l'md} = \frac{86}{39} = 2.21 \text{ (to be used in subsequent determinations).}$$

arterial pressure-time records were obtained through a small plastic catheter[1] 0.5 min. interval diameter advanced either into a subclavian artery, through a thin-walled 18-gauge needle in a brachial artery, or into the aortic arch by way of a needle in a femoral artery. The proximal end of the catheter was attached to a three-way stopcock which allowed alternate recording with a strain

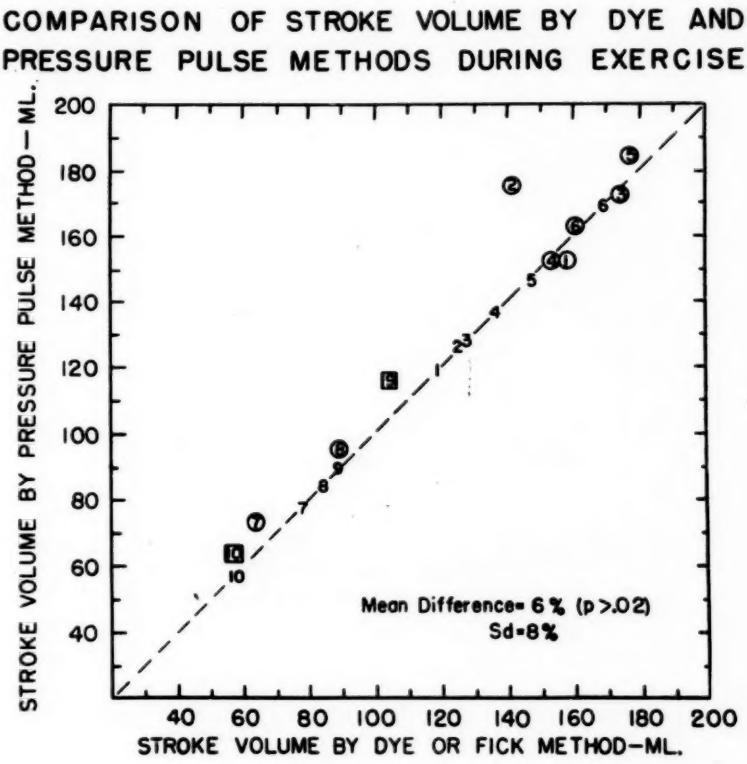

Fig. 2.

Person exercising:

Pmd = 41 mm. Hg (measured value)

Sa/Da = 0.93 (measured value)

Equation 9:

$$SV = kPmd\left[1 + \frac{Sa}{Da}\right]$$

$$= 2.21 \cdot 41\ (1 + 0.93)$$

$$= 174 \text{ cc.}$$

SV (dye) = 179 cc. (measured value).

Before showing the results of comparisons of this method with the Fick and dye method, I should like to describe briefly the techniques employed and some of the precautions taken to insure a faithful recording of pressure. Central gauge and a capacitance (Lilly type) manometer. The output of the manometers was fed into d'Arsenval type galvanometers and recorded photographically using a two meter light arm. This alternate recording arrangement was used to test the minimum response characteristics necessary to record a central arterial pressure pulse.[5]

After withdrawing the catheter at the conclusion of each experiment, the tip was placed in a low frequency oscillator chamber and the response of the system to square wave and sine wave pressure variations of varying frequency was tested. The strain gauge system was almost

optimally damped with its peak response at 15 cps while the capacitance manometer was underdamped, overshooting 700 per cent at its peak frequency of 90 cps.

pressure through indwelling needles. Dye concentration in arterial blood was recorded continuously with a curvette oximeter and an ear piece oximeter. Curves of dye concentration were

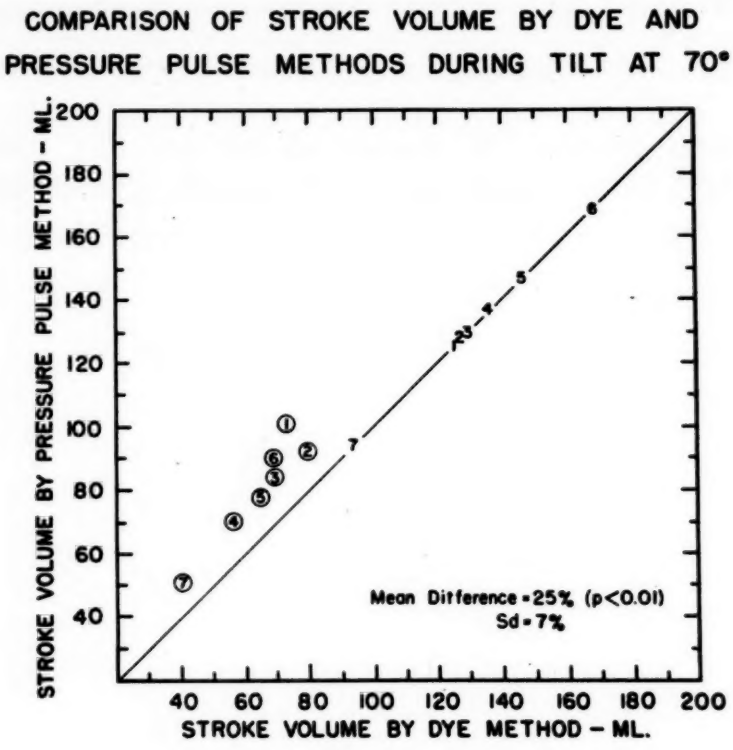

Fig. 3.

A comparison was made of aortic pressure pulses recorded by the strain gauge and the capacitance manometer-catheter systems. It is of interest that the vibrations observed, just preceding the onset of systole, and at the anacrotic and dicrotic notches, were of the resonant frequency of the catheter-manometer system, and thus do not represent actual pressure variations within the arterial system. No measurable difference in the pulse recorded by the two systems was found except in the time delay in onset as measured from the R wave of the electrocardiogram. The calculations made in the present study were carried out on recordings made with the strain gauge manometer. Because of the stability of this instrument, it was considered the manometer of choice for this type of study. Femoral artery pressure was recorded through a short catheter and right brachial and right radial artery

recorded at a camera speed of 5 mm/sec. while the pressure pulses were recorded simultaneously with another camera at a speed of 80 mm/sec. In this method I have just described a record of a central and only one peripheral pulse is required.

Figure 2 shows a comparison of stroke volume by the dye or Fick and the pressure pulse methods during exercise and includes data from six normal subjects and four patients with mitral stenosis. The uncircled numbers on the line of identity are the resting values and are shown only to demonstrate the magnitude of the change in stroke volume that occurred during exercise. The circles are comparisons made during exercise with the dye method and the squares with the Fick method. In this series the mean stroke volume increased only 11 per cent, but the average exercise cardiac output was 182 per cent of

the resting value and heart rate increased thirty-five beats per minute. The mean difference between the values obtained by the pressure pulse, and the control method was 6 per cent and the suit which applies 75 mm. Hg pressure to the entire body below the xyphoid. Under these circumstances no systematic difference was obtained and the standard deviation of the differences was

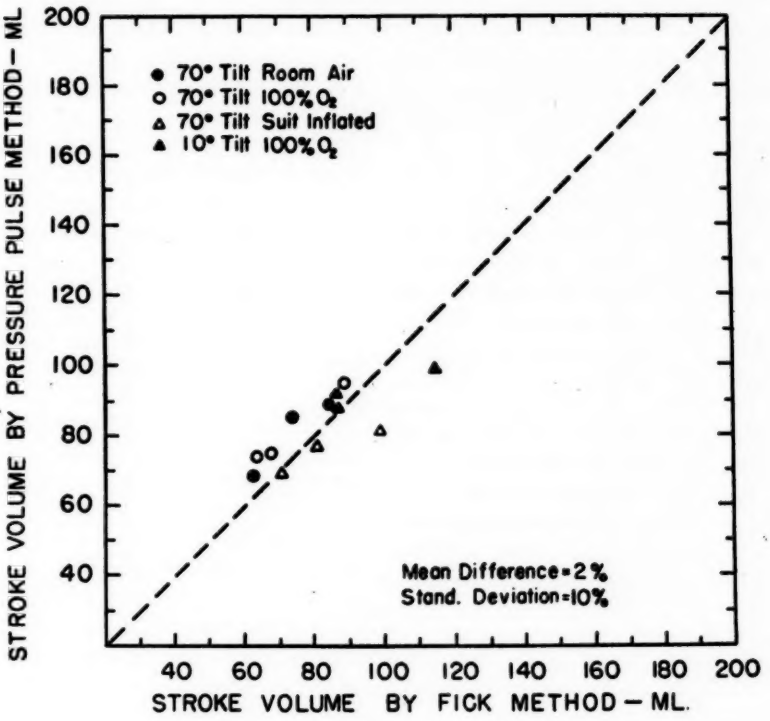

Fig. 4.

standard deviation of the differences was 8 per cent.

Figure 3 shows a comparison of stroke volume by the pressure pulse and the dye method carried out four to five minutes after tilting the subject to head up position, 70° from the horizontal. Under these circumstances a significant systemic difference was observed, but the standard deviation about this difference was small, being only 7 per cent. In the tilt position, stroke volume averaged 61 per cent of the resting value by the pressure pulse method, and 53 per cent by the dye method. The reason for the systematic difference in the tilted position is not clear as yet.

In Figure 4 are shown twelve comparisons of values for stroke volume obtained by the Fick and pressure pulse methods under a variety of physiologic conditions, including tilt to various positions and inflation of a new type antiblackout 10 per cent. In this series of observations, mean arterial pressure varied from 81 to 149 mm. Hg, and heart rate from 84 to 125 beats/mm., providing a rather rigid test of the ability of the method to follow stroke volume changes under conditions producing marked alterations in the status of the cardiovascular system.

In conclusion, a method for estimation of stroke volume in man from a recording of a central and a peripheral pressure pulse has been presented. This requires the obtaining of a factor relating change in arterial pressure to change in volume, by carrying out a determination of stroke volume during a steady state period by an independent method. Inasmuch as the usefulness of a pressure pulse method lies primarily in its ability to follow rapid changes in stroke volume, this would not appear to be a serious disadvantage. Basically this approach differs from

(Continued on Page 130)

CARDIAC CATHETERIZATION IN DIAGNOSIS OF CONGENITAL HEART DISEASE

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NOTABLE IMPROVEMENT in the diagnosis of congenital heart disease has been achieved as a result of the introduction of cardiac catheterization. The purpose of this communication is to point out information that can be obtained by this procedure as well as to indicate its shortcomings. Catheterization may provide useful data by the following means: (1) the demonstration fluoroscopically of the *introduction of the catheter* into an *abnormal pathway;* (2) the demonstration of an abnormal pressure gradient across the pulmonic or tricuspid valve, indicative of stenosis of the valve; (3) the demonstration of the identity of the systolic pressures in the right ventricle and brachial artery, indicative of an overriding aorta or a functionally similar condition; (4) the demonstration of the presence of arterialized blood in one of the chambers of the right side of the heart or in the pulmonary artery, indicative of a left-to-right shunt; and (5) the demonstration of arterial oxygen unsaturation not corrected by the administration of 100 per cent oxygen of a right-to-left shunt. The latter procedure does not involve catheterization, but is often carried out in conjunction with it.

In addition, the determination of flows, pressures, and resistances at rest and during various physiological stresses, permit a better appreciation of the abnormal physiological processes and a deeper insight into the clinical course and natural history of the disease.

1. *Introduction of catheter into or through abnormal pathways.*—This is one of the most reliable ways of diagnosing a congenital heart lesion if the abnormal pathway can be definitely identified. Catheters have been passed through or into the patent ductus arteriosus, aortic septal defects, atrial septal defects, anomalous pulmonary veins, ventricular septal defects, overriding aortas, double superior vena cavas and others. It is usually not difficult to determine the catheter's exact position with the aid of oblique and lateral as well as anterioposterior fluoroscopy. In my opinion it is, however, extremely difficult, if not impossible, to differentiate an anomalous pulmonary vein from an atrial defect, as the course pursued by the catheter may be very similar.

2. *Abnormal pressure gradient across a valve.*—There is normally no more than 1 to 2 mm. Hg difference in pressures between the right auricle and right ventricle during the first fraction of a second of diastole, or between the right ventricle and pulmonary artery, during systole. The most accurate and sensitive indication of stenosis of the tricuspid or pulmonic valve is the demonstration of a significant pressure gradient across the valve. Until 1946, only about seventy cases of pure pulmonic stenosis were described pathologically in the medical literature. Since the advent of physiological measurements, enabling pressures to be recorded on both sides of the valve, many hundreds of cases have been recognized. Pulmonic stenosis may now be considered to be one of the most common of congenital defects. I hope Dr. Bing will describe the manner in which the valvular type stenosis can be differentiated from the infundibular type by means of a cardiac catheter.

3. *Identical systolic pressure in right ventricle and brachial artery.*—With dextroposition or overriding of the aorta, the aorta and right and left ventricles are in direct communication during systole. With minimal overriding, there may be a difference in systolic pressures of the right ventricle and aorta. When free communication is present, however, the pressures are essentially the same *under all circumstances.* Brachial arterial pressure tracings in the adult have been found to be more representative of aortic pressure than femoral or radial artery recordings. In all but one of our cases with established overriding, identical brachial arterial and right ventricular systolic pressures have been demonstrated. The

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one exception had a brachial arterial pressure 20 mm. Hg lower than the right ventricular pressure; this was perhaps due to an anomaly of the arteries supplying the upper extremity.

Identical systolic pressures in the right ventricle and the aorta are also present in large intraventricular septal defects, cor triocular biatrium, and common truncus arteriosus. In these conditions, there is no anatomical overriding of the aorta, but there is free communication between the right and left ventricles and the aorta during systole. Thus identity of systolic pressures in the right ventricle and brachial artery is found in lesions with a wide anatomic variation but with a common physiological behavior.

In primary pulmonary vascular disease, or when pulmonary vascular complications have developed in mitral stenosis, ventricular or atrial septal defects, or patent ductus arteriosus, the right ventricular systolic pressure may be elevated to systemic levels and be identical with that of the brachial artery at rest. Our experience indicates the identity of such pressures to be more than mere coincidence. This group of lesions can readily be differentiated from those with free communication between right ventricle and aorta by altering the pressures with exercise or by the administration of hypertensive or hypotensive agents. In those with a free communication between right ventricle and aorta, pressures in both areas rise and fall together, whereas in the second group, the systolic pressure in the right ventricle and/or pulmonary artery rises on exercise much higher than that in the brachial artery.

4. *Left-to-right shunts.*—Left-to-right shunts can be recognized by noting the appearance of arterialized blood, that is, blood high in oxygen content, in the pulmonary artery, right ventricle, or right auricle. The interpretation of this may not be simple. For example, the presence of arterialized blood in the pulmonary artery may signify any of the following: (a) patent ductus arteriosus, (b) aortic septal defect, (c) blood aspirated from the pulmonary capillaries and veins. The latter occurs only when the catheter is wedged into a terminal branch of the pulmonary artery, and thus should not lead to the erroneous interpretation that a shunt is present. Differentiation between the first two, however, is impossible by determining the oxygen contents of pulmonary arterial blood samples.

The appearance of arterialized blood in the right ventricle indicates either: (a) ventricular septal defect, or (b) patent ductus arteriosus with pulmonic insufficiency. Usually there is no difficulty distinguishing the two by clinical means unless both defects co-exist.

Arterialized blood in the right auricle may be due to: (a) atrial septal defect, (b) anomalous pulmonary vein entering the right auricle, (c) ventricular septal defect with tricuspid insufficiency, (d) common auriculoventricular valve with shunt from left ventricle to right auricle, and (e) ? coronary auriculoventricular aneurysm. With the exception of ventricular septal defect with triscuspid insufficiency these lesions cannot be accurately differentiated from one another by cardiac catheterization. I am not sure that a coronary auriculoventricular aneurysm provides a sufficient amount of oxygenated blood to raise the oxygen content in the right auricle sufficiently to be recognized. An elevated oxygen saturation of blood withdrawn from the coronary sinus should be diagnostic. Atrial septal defect, is by far the most common of the lesions listed, and it is for this reason that this diagnosis is often made so dogmatically. I am a sinner in this regard. Actually, the above differential always exists.

Thus, abnormal physiology can be translated to abnormal anatomy only as an approximation. The precise location of the defect in the atrial, or ventricular septum, cannot be determined. The size of only the smaller defects, wherein there is a pressure gradient across them, can be calculated. The larger ones can easily be recognized, but it cannot be ascertained if it is a defect of 3 cm.^{2}, or one in which there is no septum, or only an irregular peripheral rim. These points are of extreme importance to the surgeon, but cannot be answered by cardiac catheterization.

The magnitude of the shunt can be calculated using the Fick principle. I agree with Dr. Stow and Dr. Visscher that the Fick output, under the most ideal conditions, lacks precision, and that when shunt flows in congenital heart diseases are calculated, the errors may be great. It was with considerable hesitation that we originally made such calculations, and at best they may afford only crude approximations. The figures are at times so grossly in error as to be ridiculous. We have studied two patients with presumable atrial septal defect in whom the oxygen content of the pulmonary arterial blood was the same as that taken from the brachial artery. The pulmonary

blood flow, as calculated by the Fick principle, was infinite. Our most recent case had a calculated pulmonary blood flow of 70 liters per minute. Obviously the error in calculation was so great as to be meaningless, except that it indicated a large shunt.

As discussed previously, the main errors stem from the presence of extremely narrow arteriovenous oxygen differences, and the difficulty of obtaining representative mixed venous blood samples. Depending on the reliability of these two factors, the calculation of flows, shunts, resistances, and cardiac work may or may not be justified.

Not infrequently young patients are seen with a congenital murmur, but with no disability except for cardiac anxiety of iatrogenic origin. Cardiac catheterization, satisfactorily performed, fails to detect abnormalities of pressure, oxygen content of blood samples, or calculated flow. Two possibilities exist to explain the murmur—either that there is a lesion in the left side of the heart, for example, subaortic stenosis, or that there is one in the right side of the heart which is too small to be detected by this technique. If, on consideration of all information available, the latter possibility appears to be the answer, one can then reassure the patient that the lesion, no matter what it may be, is of no clinical significance, whatsoever, and that he may consider himself to have in essence a normal heart. There is one reservation that must always be added, that is the potential vulnerability to subacute bacterial endocarditis. Of all lesions under these circumstances, the one most easily escaping detection is a small ventricular septal defect. Although small atrial septal defects are undoubtedly undetected more frequently than any other lesion, by the time they produce a murmur, their detection is usually straightforward.

5. *Right-to-left shunts.*—In general, angiocardiography is a much more valuable method for identification and delineation of right-to-left shunts than cardiac catheterization. Right-to-left shunts are recognized by the failure of arterial blood to become fully saturated upon the administration of 100 per cent oxygen. However, the location of the right-to-left shunt cannot be determined by this method, although this can often, but not always, be accurately deduced by noting that a given chamber of the right side of the heart has a higher pressure than the corresponding chamber of the left side. Actually such studies are poor in the precise information they give, and have been greatly improved by the ether test of Soulié, and especially by the dye injection technique of Wood, which will be discussed by Dr. Swan in the next communication.

6. *Understanding of abnormal physiology.*—Perhaps the most important contribution of cardiac catheterization to congenital heart disease, is the knowledge that is being acquired with regard to the pathological physiology of the circulation in the different types of lesions. In the past, congenital heart disease has been studied mainly from the anatomical and embryological points of view. The physiology and clinical aspects are currently under intensive study. As corrective surgery becomes more and more applicable to the various lesions, such studies will increase. There are now considerable gaps between the anatomical, physiological, and clinical interrelationships of the various congenital lesions. These must, and will be, filled in in the years to come. What, for example, is the clinical course of patients with atrial septal defect? I do not know the answer clinically or physiologically. Cardiac catheterization, with all its handicaps and limitations, serves as the single most important physiological tool of approximate diagnosis and investigation. Other physiologic tools must be added to bridge some of the many gaps that exist in our knowledge of these diseases.

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Discussion

DR. R. J. BING, Baltimore, Md.: I would like to comment briefly on two problems. One, the relative diagnostic value of special tests as compared to clinical procedures, and secondly, some of the problems of pulmonic stenosis with particular reference to its surgical correction.

There has been a great deal of discussion on the relative value of clinical methods, versus special tests in congenital heart disease. Most of us agree, that in a great majority of patients with congenital malformations of the heart, a diagnosis can be made by clinical means. I use the term "clinical" very loosely, because it comprises not only the history and physical examination, but also electrocardiography and fluoroscopy.

Special tests are catheterization of the heart and angiocardiography. I would like to mention here some of the specially helpful points in the clinical diagnosis of congenital heart disease. As to history: repeated respiratory infections suggest the presence of increased pulmonary blood flow and/or pulmonary hypertension; anoxic convulsions are in line with a marked diminution in pulmonary flow, and hemoptyses suggest pulmonary hypertension. If a non-cyanotic child is reported to be dyspneic on slight exertion, the possibility of isolated valvular pulmonary stenosis must be entertained.

The most important point, as regards to physical examination, is absence or presence of cyanosis. If cyanosis is present, then a right-to-left shunt exists. But since a right-to-left shunt can exist in patients who have diminished pulmonary artery pressure, and in those in whom it is elevated, other physical signs of diminished or increased pulmonary pressure and flow must be looked for. Thus, a marked accentuation of the second pulmonic sound points toward pulmonary hypertension. The location and character of the murmur, although not as important as in acquired heart disease, may give valuable information. If a systolic murmur is present in a non-cyanotic child, high in the second intercostal space to the left of the sternum, a valvular pulmonic stenosis may be the underlying malformation. Absence of a murmur in a severely cyanosed child suggests pulmonary atresia.

It may be seen from these isolated examples, that a good deal of information may be gained from conventional methods. Add to this the information gained from electrocardiography, and particularly from fluoroscopy, and it is little wonder that one is often able to arrive at a correct diagnosis by conventional methods alone.

Why, we must ask ourselves, do we then in many cases resort to the difficult procedures of catheterization and angiocardiography? Is it that in complicated malformations, clinical examination is not sufficient? This we believe not to be correct. It has many times been our experience that the diagnosis of complicated malformations has been made more difficult by catheterization and angiocardiography. Exact location of the catheter tip in the heart, is often debatable in these patients, or the contrast material may have entered a portion of the heart which cannot be accurately defined. On the other hand, special tests are often essential in diagnosing the more common malformations of the heart, particularly if there is overlapping of signs and symptoms. This is particularly the case in the non-cyanotic group. Frequently the differential diagnosis between auricular, ventricular septal defect, or patent ductus arteriosus is difficult without catheterization, particularly in the absence of typical murmurs. In the cyanotic group it may be difficult to differentiate a tetralogy of Fallot with large collateral circulation from an Eisenmenger's Complex or true truncus.

I would like to say a few words about the problem of pulmonic stenosis. Anatomically, it is not an isolated malformation, rather the type of associated malformation that influences the physiologic pattern, and therefore the surgical correction of the stenosis.

When pulmonary stenosis is associated with a ventricular septal defect, the stenosis is usually of the infundibular type. In the presence of an auricular defect, the stenosis is usually at the pulmonic valve. If there stenosis is usually at the pulmonic valve too. When is neither auricular nor ventricular septal defect, the there is overriding of the aorta, such as in the tetralogy of Fallot, the infundibular type is frequently found in the tetralogy of Fallot.

In infundibular stenosis, the subpulmonary tract is bounded by the right ventricular muscle, by the ventricular septum and by a muscle which separates the tract from the ventricular septal defect. This latter muscle is the crista supraventricularis. This subpulmonary tract may at times be very narrow and it is believed by some, including Blalock, that this subpulmonary tract is the primary source of obstruction in the infundibular type. Brock, on the other hand, while admitting that the infundibular tract is often hypoplastic, maintains that as a rule an effective lumen is adequate, and that the actual stenosis occurs at one level and is linear and diaphragmatic.

This difference in interpretation is of considerable surgical treatment. If the subpulmonary tract has an effective lumen and if the stenosis is linear, infundibulotomy may be practical; if the stenosis is a long narrow channel, this procedure is hardly adequate, and even infundibular resection may be difficult.

We examined this question by means of drawings and wax casts, which were prepared by pouring parafin into the cardiac chambers and thus obtaining a mold of the cardiac cavities. By means of these casts and drawings, we could show the reverted funnel-shaped entrance into the subpulmonary tract of the right ventricle, and we were also able to show that in addition to the narrowing of this tract resulting from hypertrophy of the muscle of its posterior wall producing "muscular stenosis;" there is also a secondary stenosis or "fibrous stenosis" which is linear and diaphragmatic and which is composed of fibrous rather than muscular tissue. It is our opinion that the fibrous stenosis results in very little additional narrowing of the internal diameter of the subpulmonary tract. Therefore, infundibular resection is the only direct surgical approach; whether this is able to overcome the obstacles, by removing a mass of muscular tissue of the subpulmonary tract, is still a very controversial question.

In valvular pulmonic stenosis, the morbid anatomy is very different, as has been shown by Edwards et al. Although there is often muscular hypertrophy, this does not result in narrowing of the right ventricular outflow tract. It is not surprising, therefore, that valvolotomy is the operation of choice in this malformation.

The physiopathology of these malformations have been described in great detail, and Dr. Dexter has mentioned most of the essential features. I want to mention only the physiologic and surgical importance of the degree of overriding of the aorta, in the surgical correction of pulmonic stenosis. It has been shown that most patients in whom the aorta overrides more than 50 per cent, died, during or shortly after construction of an artificial ductus, from acute pulmonary edema or severe pulmonary congestion. On the other

hand, if there is no overriding, the construction of an artificial ductus results in right sided failure. The reason for this is that when there is marked overriding, the increased volume of blood reaching the left ventricle from the lungs, often this construction of the ductus has no escape. In the presence of pulmonic stenosis and an auricular defect, where there is no overriding, the construction of an artificial ductus leads to right side failure because increased return of blood into the left auricle will raise the left auricular pressure, and by tending to equalize the gradients between right and left auricle, diminish the right-to-left shunt. The subsequent increase in right auricular pressure leads to failure. Special tests have contributed very little to the recognition of the degree of overriding of the aorta. However, in Sweden, the use of regional angiocardiography has shown the way.

Physiologic tests have been particularly useful in the interpretation of operative procedures for pulmonic stenosis. In the tetralogy of Fallot, the construction of an artificial ductus produces a postoperative rise in the oxygen content of right ventricular blood. This may be due to increased inter-cardiac left to right shunting. This increased left to right shunt may also be responsible for the elevation of the right ventricular pressure which was observed in many patients.

In the treatment of isolated pulmonic stenosis with auricular defect, a condition in which valvolotomy is the method of choice, there is a fall in right ventricular pressure, which, however, never returns to normal; the pulmonary blood flow increases and the peripheral oxygen saturation rises.

Physiologic data obtained before and after infundibular resection in the tetralogy of Fallot are still scarce. However, Bailey and Glover used the infundibular resection in forty-two patients with the tetralogy of Fallot; of these twenty-two had infundibular stenosis. In patients who were catheterized postoperatively, the gradient between the systolic pressure in the right ventricle and the pulmonary artery was still very marked; the clinical results on the other hand, were far more satisfactory than one might expect from these physiologic data. It is hoped that future physiologic studies will justify some of the optimism evoked by these clinical observations.

Dr. H. B. Burchell, Rochester, Minn.: In respect to the general principles outlined by Dr. Dexter pertaining to cardiac catheterization and its diagnostic application, I am in agreement, although there are a number of small points about which I have somewhat different opinions. I can re-emphasize the difficulty in the differentiation of atrial septal defect and anomalous pulmonary venous connection (drainage) into the right atrium. Actually, no true differentiation can be made when the posterior segment of the atrial septum is missing.

I should like to reiterate the diagnostic value of exercise, not only for patients who have ventricular septal defect, but also for persons who have pulmonary stenosis. The obstructive nature of pulmonary stenosis, with an intact ventricular septum is often dramatically portrayed after exercise, when the right ventricular pressure may be increased much further while the cardiac output is insignificantly increased. Like Dr. Dexter, I consider that similarity of right ventricular systolic and systemic systolic pressures, particularly when this pressure relationship persists with exercise, is a strong indication of a large ventricular septal defect. It is important to remember, however, that the actual values need not be identical; systolic pressures in the radial artery that are 10 to 25 mm. of mercury in excess of the right ventricular systolic pressure can be regarded as "physiologically" identical. While in general it is considered that the right ventricular systolic pressure is correlated with the size of the ventricular septal defect, exact correlations in this regard are not available.

Dr. Dexter mentioned that breathing 100 per cent oxygen would differentiate cyanosis due to an intracardiac shunt from that due to pulmonary disease. It is important to recognize, that exceptions to this conclusion occur in that a right-to-left shunt, that is present when the patient is breathing air, may disappear when the patient breathes 100 per cent oxygen. This is thought to be related to a decrease in pulmonary resistance. A number of such instances have been encountered in which right-to-left shunts are demonstrable by dye techniques when the patient breathes air but which disappear when 100 per cent oxygen is used. In such cases the arterial oxygen saturation may be 100 per cent and the blood may carry its normal complement of dissolved oxygen. Previous papers in this symposium have pointed out the potential errors in the determination of pulmonary flow using the Fick principle; Dr. Dexter has placed proper emphasis on this problem. This is something that must be remembered when we calculate pulmonary resistance with high pulmonary flows, and we must avoid drawing too definite conclusions regarding the change in resistance following the cure of large left-to-right shunts.

Any physician, intimately concerned with the diagnostic problems presented by patients who have congenital heart disease, probably will admit two things: (1) his clinical diagnostic ability has been increased many fold in the past ten years as the result of correlation of clinical findings and data derived from cardiac catheterization, and (2) in an occasional baffling case, the results of cardiac catheterization may be diagnostically inconclusive or even misleading.

One principle needs constant repetition and reemphasis. In the diagnostic approach all the facts in the history and examination should be carefully sifted and weighed; any single laboratory finding that superficially appears to be definitely diagnostic should be studied with care if it does not appear to fit with the other features of the case. As an example, the finding of arterialization of blood in the left atrium might be due to many causes, including: (1) sampling error, including difficulties related to differences in the saturation of the three main veins emptying into the right atrium, namely, the superior and inferior vena cava and the coronary sinus; (2) anomalous pulmonary venous connections; (3) ventricular septal defect, with the right-sided opening above or through the tricuspid valve, this shold be suspected after the occurrence of

subacute bacterial endocarditis in patients who have ventricular septal defect; (4) rupture of an aneurysm of the sinus of Valsalva into the right atrium, and (5) persistent atrioventricular canal with a "low" atrial septal defect as well as the atrial septal defect itself.

Two procedures that have been introduced into the technique of cardiac catheterization and brought to a state approaching technical perfection by Dr. Wood and his associates, have added a great deal to the diagnostic accuracy of cardiac catheterization.

The first is the use of the oximetric curvette to give nearly instantaneous readings of oxygen saturation of the blood at the tip of the catheter. This allows many determinations to be made; in addition, continuous sampling during withdrawal of the catheter from one part to another permits small differences in the oxygen saturation between such parts to be validated.

The second procedure is injection of dye into various parts of the central cardiovascular structures. This has had the rewarding results of fairly reliable localization and quantitation of venous arterial shunts.

Certain concepts presently held concerning four of the most common congenital malformations may be of some interest:

A. *Atrial septal defect.*
   1. Preferential drainage of the right lung to the right atrium exists in the majority of cases.
   2. Slight modifications of bilateral shunting are demonstrable by postural and respiratory maneuvers.
   3. Preferential drainage of the inferior vena cava toward the left atrium often is demonstrable.
   4. Functional openings of the foramen ovale with right-to-left shunting may occur after pulmonary embolism or pulmonary disease.
   5. Easy catheterization of the left ventricle through an atrial septal defect may be suggestive of the partial form of a common atrioventricular canal (auriculoventricular commune).
   6. In anomalous pulmonary venous connections, one should also suspect the presence of an atrial septal defect.

B. *Ventricular septal defect.*
   1. Many "transitional" conditions exist between uncomplicated ventricular septal defect and cyanotic Eisenmenger's complex.
   2. Severe pulmonary hypertension may coexist with a left-to-right shunt (pulmonary blood flow greater than the systemic) without hypoxemia.
   3. Cardiac failure in early infancy is not infrequent and may be associated with clinical evidence of left ventricular enlargement.
   4. Diastolic shunting of blood may be an important avenue of investigation.

C. *Patent ductus arteriosus.*
   1. The time of appearance of the continuous murmur is variable.
   2. Evidence of associated defects, particularly coarctation of the aorta and ventricular septal defect, is to be searched for.
   3. A patent ductus arteriosus is to be considered as the possible cause of cardiac failure appearing in early infancy.
   4. The disease in adult patients who do not have continuous murmurs and "reversed" shunts may be of two types: (a). An unchanged pattern since infancy, with no evidence of previous left-to-right shunt and no cardiac enlargement. (b). Acquired pulmonary hypertension and true reversal of shunt, which often may be exaggerated by breathing mixtures that are low in oxygen.

D. *Pulmonary stenosis.*
   1. Many difficulties in the evaluation of the subpulmonic tract remain, even though the problem has been approached through cardiac catheterization, blind surgical exploration and anatomic dissection.
   2. Extremely large hearts, as manifested by their roentgenologic silhouettes, may be present in infants who have isolated pulmonary stenosis.
   3. Surgical exploration sometimes deserves precedence over cardiac catheterization.
   4. Left-to-right shunts (pulmonary blood flow greater than the systemic) at either atrial or ventricular levels may coexist with severe pulmonary stenosis.
   5. Mild pulmonary stenosis is a definite entity, although its anatomic basis is obscure.

Time does not allow discussion of the difficult diagnostic problems often presented by the newborn or young cyanotic infant or the difficulties in the interpretation of murmurs, electrocardiographic records or roentgenologic findings. This often is best done by presentation of cases. This symposium has, I think, been properly oriented toward the discussion of broad concepts and of accurate measurment of hemodynamic variables.

In closing, it is proper to pay tribute to Dr. Cournand, who demonstrated the relative safety and value of cardiac catheterization in man, but to emphasize that this procedure does present some risk to the patient, the mortality rate probably approximating one death in 500.

DR. PAUL ADAMS, Minneapolis, Minn.: At the University of Minnesota, we have been particularly interested in what we consider the most challenging and difficult problem in the differential diagnosis of congenital heart disease, that is, infants under two years of age.

Challenging, because next to prematurity, it has been the largest single cause of infant mortality in Minnesota. Review of our autopsy material reveals that a certain number of these patients had defects which could have been corrected or improved by present surgical techniques. An ever-increasing variety of malformations may soon be similarly considered.

We consider it difficult because, in our experience, symptoms and findings in the infant differ markedly from the classical descriptions when malformation is severe. We are gradually becoming aware of findings, which are fairly typical of each individual malformation in the infant. We have often been asked, and indeed occasionally ask ourselves, just how much is accomplished by these special procedures in the infant, compared to the added risks involved? With that question in mind, our recent experience with heart catheterization in infants will be briefly outlined.

Right heart catheterization has been performed in fifty-three infants under two years of age—about 40 per cent of the total number of pediatric catheterizations during 1952-1953. Angiocardiography and retrograde

aortography were done on seventy-four infants during the same period. These special procedures were undertaken only when the infants were considered to be severely handicapped, when the risk of death during infancy appeared to be greater than the risk of special procedures, and when the specific type of malformation could not be diagnosed by routine methods. The smallest patient weighed only 3 kilograms at four months of age. Of the total of 127 special procedures (heart catheterization, angiocardiography and retrograde aortography, one death occurred during heart catheterization. This death was probably related to the medications used rather than the procedure, *per se*.

The final diagnoses of the fifty-three patients who had heart catheterization studies are listed as follows:†

1. Patent ductus arteriosus	6
2. Coarctation of the aorta	3
3. Interatrial septal defect	3
4. Interatrial plus interventricular or A-V canal	4
5. Anomalous pulmonary venous drainage into the right atrium	5
6. Interventricular septal defect or Eisenmenger's	13
7. Tetralogy of Fallot	7
8. Transposition of the great vessels	5
9. No left-to-right shunt	
(a) aortic stenosis	4
(b) endocardial fibrosis	3

Patent ductus arteriosus is the malformation in which the greatest salvage has been obtained. All patients had been severely ill with respiratory infection, and/or growth retardation, and their survival had been considered doubtful. None of these infants had the typical continuous murmur. The location and character of the systolic murmur only occasionally was suggestive of the patent ductus ateriosus. Surgical closure of a very large ductus was successfully accomplished in every case with marked improvement. One diagnostic error occurred in which the oxygen saturations of the pulmonary artery were increased over the remaining venous samples. However, at surgery and at post mortem a high interventricular defect alone was found. This occurred before we were using retrograde aortography. Since that time we have used that method successfully in three additional cases.

Three out of a total of ten infants, who were operated on for coarctation of the aorta, were considered to need heart catheterization studies prior to surgery because of the suspicion of serious intracardiac defects. Although surgery was not denied any of these patients, a more guarded prognosis could be given.

The "flush" method for determination of blood pressure has been most helpful in infants. Occasionally, when the infant is crying or struggling during the examination, an unusually high blood pressure has been found in the arms in normal infants but normal pressures in the leg when done subsequently. To avoid this possibility of error in young infants we have simply joined two infants' cuffs together with a Y tube connector and taken pressures of the arm and leg simultaneously by the "flush" method. In the normal infant without coarctation, the leg flushes slightly before or simultaneously with the arm. In coarctation the flushing of the leg is delayed.

Transposition of the great vessels and tetralogy of Fallot can best be diagnosed by angiocradiography.

Although seven patients had no demonstrable left to right shunts, we feel that they were valuable in localizing the lesion to the left side of the heart. Two patients thought to have endocardial fibrosis clinically before catheterization, were shown to have patent duc'us arteriosus as a result of catheterization.

The results of our diagnostic efforts have in many instances been encouraging, especially in those infants where excellent surgical correction has been accomplished. At times we have been disappointed, but overall, we feel that these sick infants deserve every effort of diagnosis that we can make.

We have felt that valvular pulmonary stenosis could be easily distinguished from infundibular stenosis by the characteristic sharp change in pressure at the valve in the valvular type, and the slower transitional change in the infundibular type. On two occasions, however, this sharp transition occurred and at thoracotomy they were found to have an infuidibular type. Both of these patients showed a small left to right shunt in the right ventricle in addition.

I should like to ask (a) whether this experience has occurred elsewhere? (b) if so, what explanation can be offered as to why it occurs, and (c) is there a way that this error may be avoided in the future?

Dr. Dexter mentioned the use of certain drugs and exercise as a differential aid in cases of suspected pulmonary hypertention, as opposed to the aorta arising from the right ventricle.

We have had one death in a patient with pulmonary hypertension following fluorescein circulation time, and recently the *New England Journal of Medicine* has reported such a case with mortality following a decholin circulation time. I would like to ask Dr. Dexter if he feels that the use of drugs or exercise in such a situation might be considered to add more risk to the procedure of heart catheterization than they care to undertake.

Dr. Dexter stated, if I understood him correctly, that in the absence of a significant shunt as shown by oxygen saturations during a satisfactory heart catheterization, any defect that existed could be safely considered as clinically insignificant.

An exception to this, I believe, might occur in a high interventricular septal defect with pulmonary hypertension, at a certain point where the right ventricular hypertension was balanced with the left so that no left to right or right to left shunt occurred, and yet the defect obviously would be clinically significant.

†It should be stressed, as Dr. Dexter stated, that heart catheterization was only one factor in the diagnosis. The patient's history, physical examination, x-ray, angiocardiogram and retrogradeaortogram were occasionally necessary. In some instances, the correct diagnosis was learned only during postmortem examination.

DIAGNOSTIC APPLICATIONS OF INDICATOR DILUTION CURVES IN HEART DISEASE

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AN INDICATOR dilution curve shows the changing concentration of an indicator at a specific point in the vascular system at various times following its injection at some different point in the vascular system. Usually dilution curves of the indicator are obtained from the arterial system following rapid injection of the indicator into the venous system proximal to the pulmonary capillaries. An indicator substance should possess the following qualities: (1) The substance must be nontoxic and sterilizable in regard to pathogenic organisms. (2) The concentration of indicator should remain relatively constant for at least a short time and preferably should allow for storage over several days. Otherwise a supply of fresh indicator must be readily available. (3) Injection of the substance must be possible in a brief time and this injection must not cause cardiovascular responses. Thus the volume in which the indicator is contained must be small. (4) The indicator must not pass out of the vascular system in appreciable amounts for several minutes. (5) It must be possible to measure the concentration of indicator in blood or plasma with considerable precision.

The indicator in most common use is Evans blue dye (T-1824),[2,4,8] but saline solution was used by Stewart, and brilliant vital red and other dye substances by Hamilton and associates,[3] in their fundamental studies on the problem of dilution of an indicator by the circulation. Hamilton and associates[3,4] used a discontinuous method of sampling in which multiple small samples of blood were collected, the concentration of T-1824 in plasma was determined photometrically, and a concentration-time curve was constructed. From such curves it is possible to measure circulation times and to estimate cardiac output and central blood volume. But when the information to be derived from a dilution curve depends to a major extent on the contour of that curve then use of a continuous recording instrument would appear to be essential. The oximeter[13] and densitometer[6] are instruments which permit the continuous recording of the concentration of T-1824 in whole blood. However, fluctuations in the oxygen saturation of arterial blood which frequently occur in patients with cyanotic congenital heart disease will distort dilution curves of T-1824 recorded by these instruments. This serious disadvantage which is inherent in the oximetric technics renders certain radioactive isotopes attractive as potential indicators when problems pertaining to dose and recording devices are satisfactorily solved.

The observations to be discussed in this communication are based entirely on dilution curves of T-1824 recorded by means of the oximeter.

The pattern of dilution in a normal subject following injection of T-1824 into a peripheral vein is shown in Figure 1. The oximeter responds to decreasing transmission of light and hence to increasing concentration of dye by a downward deflection of the recording beam. A short interval, the appearance time, elapses between the injection of the dye and its first arrival at the recording instrument. Then there is a rapid increase in concentration to a peak, followed by a less rapid decline in concentration which is incomplete and merges into a second peak of concentration which is caused by return to the sampling site of dye which has passed the systemic capillaries.

After injection into a peripheral vein the following average normal values in seconds have been obtained: appearance time 14 (10 to 20); maximal concentration time 25 (16 to 35); buildup time 10 (7 to 15); disappearance time 16 (9 to 26) and recirculation time 21 (16 to 28).*

The most important physiologic factors modifying the dilution pattern are: the rate of flow through the system, the effective mixing volume of blood in the central vascular pool, the variation in volume and rates of blood flow in the blood vessels in the lungs, and the characteristics of flow in the aorta and blood vessels leading to the sampling site. As a rough generalization, an in-

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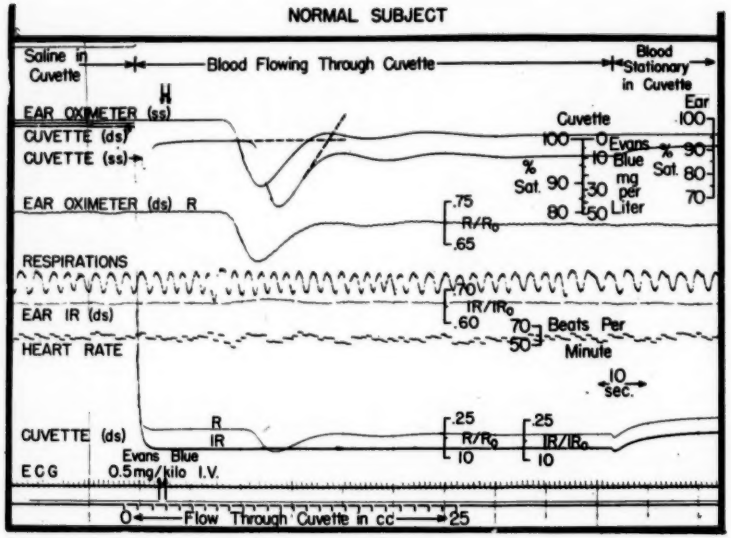
*Approximated values from Nicholson, Burchell and Wood.



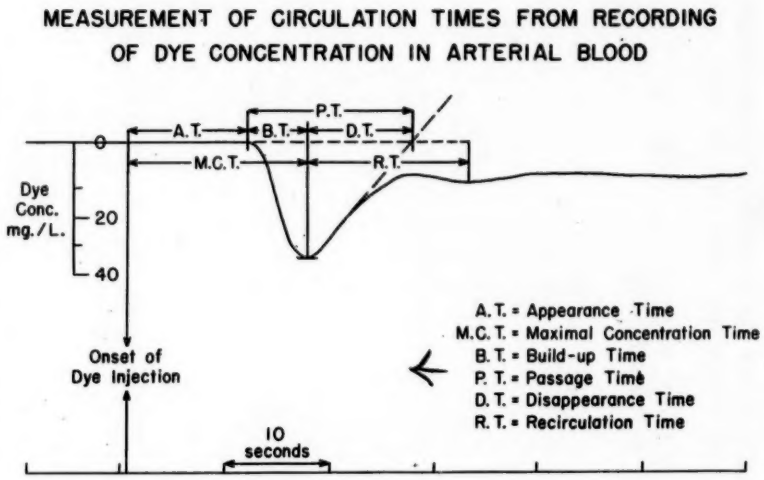


Fig. 1. Arterial dilution curves of Evans blue dye (T-1824) in a normal subject.

Left panel. A section of the photographic record, which is untouched apart from addition of captions. Curves were obtained from an oximeter on a heat-flushed ear, and from a cuvette oximeter through which blood from the radial artery was flowing. This panel shows recordings of the respiration rate, the heart rate, electrocardiogram, and rate of blood flow from the radial artery through the cuvette oximeter; also the change in transmission of red (R) and infrared (IR) light recorded by the oximeter (ds).

Right panel. The time components of the dilution curve are indicated.

crease in the volume of blood between the site of injection and the site of sampling causes a reduction in the concentration of dye, whereas a decrease in the rate of flow through the system results in prolongation of the time components. Therefore, in acquired heart disease, the changes in the dilution pattern found are those associated with reduced cardiac output and perhaps an increased volume of blood in the heart and great vessels.

In those forms of congenital heart disease characterized by an alteration in the circulatory pathway, the contour of the dilution curve may permit interpretations which have frequently been found to be of diagnostic value.

Conditions characterized by right-to-left or left-to-right shunts usually give rise to typical dilution patterns.[7] A dilution curve, following injection of dye into a peripheral vein of a patient with tetralogy of Fallot, is shown (Fig. 2, *left panel*). A shortened appearance time and an abnormal (first) hump on the buildup slope of the main curve are apparent. This pattern of dilution may be explained by the passage of a portion of the dye across the ventricular septal defect directly into the systemic circulation. The appearance time is reduced because this shunted dye reaches the recording instrument before the dye which traverses the longer, normal circulatory path through the lungs. As each portion of dye is subjected to dilution in its passage through the heart and the blood vessels, its arrival in the peripheral circulation takes the form of two curves which partially overlap. In many patients with right-to-left shunts the cardiac output may be normal, but because some dye passes through the defect, the maximal concentration is reduced from that in the blood of the normal subjects. Early in our experiences with dilution curves obtained from patients with intracardiac shunts, it became apparent that the contour of the dye curve is determined largely by the volume of shunt passing through the defect. Thus, in patients with severe cyanosis, the initial (abnormal) deflection was found to be as large or larger than the normal component of the curve, while a very small initial break was found in patients with very small right-to-left shunts producing only a minimal depression of arterial oxygen saturation. If certain assumptions are made, the magnitude of the shunt can be calculated from such curves,[12] and satisfactory agreement has been shown between shunt values calculated from the dilution curve and data obtained from cardiac catheterization. There was a standard deviation between the methods of $\pm$ 7 per cent of systemic blood flow in shunts ranging from 51 to 8 per cent of the systemic flow.

In left-to-right shunts also the dilution of indicator is altered in a specific manner[7] (Fig. 2, *right panel*). The appearance time and the buildup time are normal. However, the magnitude of the deflection is reduced, and the slope of declining concentration of indicator is much prolonged. Also, no peak due to systemic recirculation can be identified. This curve, which is typical of left-to-right shunt of considerable magnitude, is explained on the basis of recirculation of dye in the heart and vessels of the lungs with a slow clearance of a constant proportion of the recirculating dye into the systemic arterial system. This slow elimination of dye results in the prolonged disappearance time which is the most notable feature of the curves. Since, in normal subjects, sharpness of individual dilution curves varies considerably, yet the curve remains consistent with regard to internal time relations, the ratio of disappearance time to buildup time of abnormal curves provides a sensitive index of disproportionate prolongation of the disappearance time. Broadbent recorded dilution curves following peripheral injections of dye into sixteen patients with left-to-right shunts and compared the ratio of disappearance time (DT) to buildup time (BT) with the magnitude of shunt calculated on the basis of values of oxygen saturation obtained at cardiac catheterization. An approximate relation based on these data indicated that the arteriovenous shunt expressed as per cent of the pulmonary artery flow equals $10 + 10$ (DT/BT).

These dilution patterns then are typical of right-to-left and left-to-right shunts, respectively. They are dependent not only on the existence of a defect but more fundamentally on direction and volume of blood flow across it. Abnormal dilution patterns will not be seen when the pressures on either side of a defect equal one another at all times, because in this instance no shunting of blood will occur.

Since the altered dilution pattern in right-to-left and left-to-right shunts depends on the presence of an abnormal circulatory path, it appeared

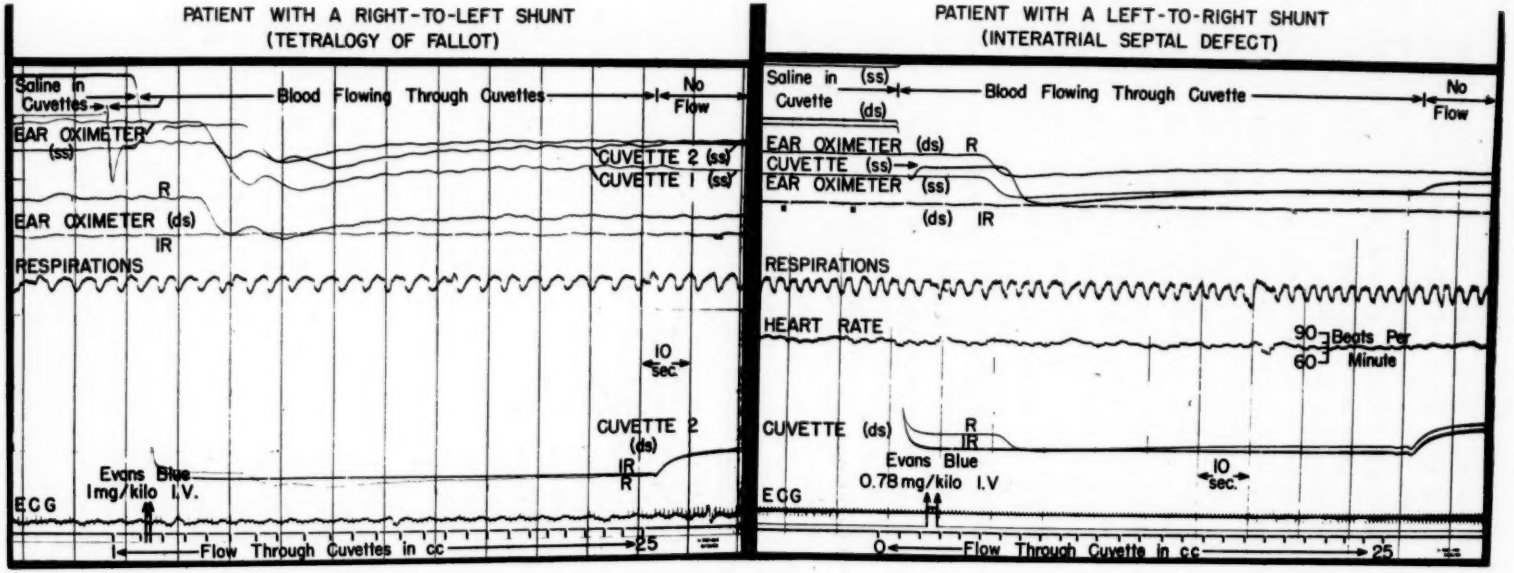


Fig. 2. Recording of dye dilution curves obtained after peripheral injection of T-1824 into a patient with tetralogy of Fallot *(left panel)* and in a patient with an atrial septal defect *(right panel)*.

Left panel. The dilution curves were recorded by an oximeter on the heat-flushed ear (ear oximeter ss) and by two cuvette oximeters in tandem (cuvette 1 ss and cuvette 2 ss) through which blood from the radial artery was flowing. The short appearance time (8.5 seconds for the ear oximeter) and the double-hump pattern seen on all curves should be noted. The normal peak due to recirculated indicator is absent. The changes in both cuvette curves were delayed due to the time necessary for dye to pass from the radial artery to the photosensitive portion of the cuvette oximeters. Small fluctuations may be noted in the recording line due to variations in oxygen saturation coincident with respiration. This form of dilution curve is typical of right-to-left shunt.

Right panel. The dilution curves from a patient with an atrial septal defect recorded by means of an earpiece oximeter (ear oximeter ss) and by a cuvette oximeter (cuvette ss) through which blood from the radial artery was flowing. The normal appearance time (11 seconds for the earpiece), the reduced magnitude of the initial deflection and the marked prolongation of the disappearance slope of the dilution curve should be noted. This type of curve is typical of left-to-right shunts.

possible to obtain additional information from dilution curves recorded after injection of dye at several different sites in the heart and great vessels of the same individual.[11] In normal subjects, is made distal to the site of a defect then a relatively normal pattern of dilution is obtained, because dye traverses a fundamentally normal circulatory pathway. But if the injection is made at or

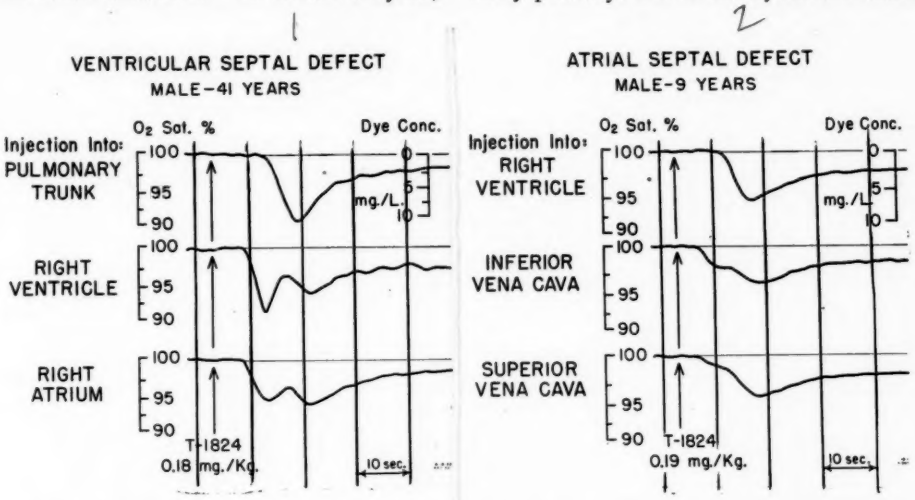


Fig. 3. Localization of the site of a right-to-left shunt in a patient with a ventricular septal defect, and in one with an atrial septal defect. In each case the curves have been superimposed in regard to the time of dye injection. The oxygen saturation scales serve to indicate the relative sensitivity of the oximeters.

Left panel. Dilution curves from a patient with Eisenmenger's complex recorded after injection of T-1824 into the pulmonary trunk, right ventricle and right atrium. Fluctuations in oxygen saturation may be noted in all curves. The appearance time after injection of dye into the pulmonary trunk is normal for this site (9.0 seconds) and the remainder of the curve is not grossly distorted. The curves after injection of dye into the right ventricle or the right atrium differ from the curve after injection in the pulmonary trunk in that the appearance time is shorter (5.5 seconds) and there is a well-defined double-hump pattern. This series of curves localizes the site of the right-to-left shunt and, hence, of the intracardiac defect to the ventricle.

Right panel. Curves obtained after injection of dye into the right ventricle, inferior vena cava and superior vena cava in a patient with atrial septal defect. The curve after injection of dye into the right ventricle has a normal appearance time (8 seconds) and build-up slope, but the disappearance time is prolonged. This prolongation of disappearance time indicates the presence of a left-to-right shunt. In the curves obtained after injection of dye into both inferior and superior venae cavae, the appearance time is reduced (4 seconds) and a double-hump pattern is apparent. In this instance, the defect can be localized to the atrial septum. The right-to-left shunt from the inferior vena cava is indicated by the curve being of greater relative magnitude than that from the superior vena cava.

and in patients without intracardiac or extracardiac shunts, the dilution pattern following injection of dye into the pulmonary trunk differed somewhat from that following injection at a more peripheral site. After peripheral injection the concentration components were reduced, and the time components were prolonged. This phenomenon was explained on the basis of greater longitudinal mixing and dilution after injection into the more peripheral vessels.[5] The curves were, however, similar to one another in general contour. In cases with right-to-left shunts it became possible to localize the site of a defect with relative certainty (Fig. 3). If an injection of dye proximal to the defect then some of the dye will be shunted through the defect while the remainder traverses the pulmonary vascular bed, thus producing the abnormal "double-hump" pattern. By this technique right-to-left shunts occurring through a patent ductus arteriosus, ventricular and atrial septal defects have been localized and, in many instances, identification would not have been made otherwise. It has been possible to recognize small right-to-left shunts, particularly those occurring at the atrial level, which caused an insignificant desaturation of the systemic arterial blood.

In cases with left-to-right shunt recirculation

of dye occurs after the dye has passed the pulmonary capillaries, hence specific differences between injections into the pulmonary trunk and produced when a portion of this shunted dye passes out (mostly by way of the left pulmonary artery) to the systemic arterial circulation. The

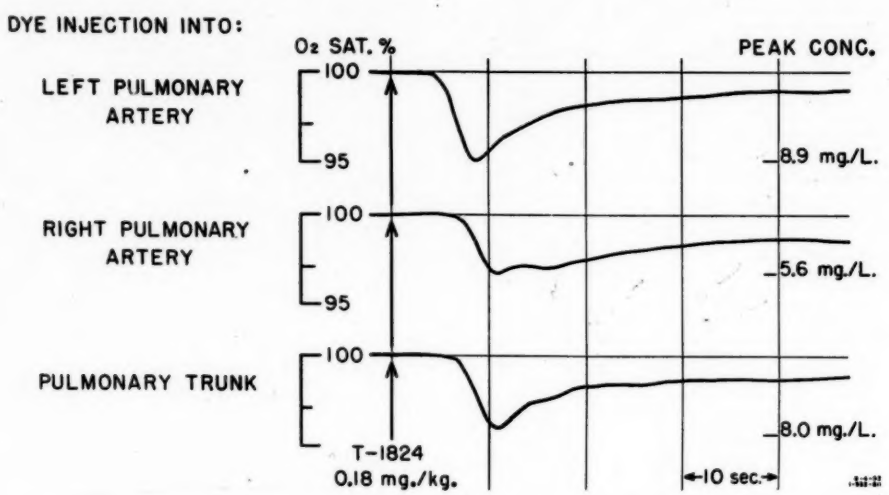


Fig. 4. Dye dilution curves obtained after injection of T-1824 into the left pulmonary artery, right pulmonary artery and pulmonary trunk of a sixteen-year-old girl with atrial septal defect. Injection of dye into the left pulmonary artery resulted in a dilution curve with a normal appearance time and build-up slope, but with a prolonged disappearance time, indicative of a moderate degree of pulmonary recirculation. After injection of dye into the right pulmonary artery, however, the magnitude of the initial deflection is reduced, and there is a well-developed second hump on the disappearance slope. The curve following injection into the pulmonary trunk is a composite of both right and left pulmonary arteries (for discussion see text).

more peripheral sites would not be expected. In a relatively limited experience with the central injection technic in patent ductus arteriosus and ventricular septal defect no remarkable distinguishing features have been apparent. However, in both atrial septal defect and anomalous pulmonary venous connection, striking differences in the dilution pattern have been obtained after injections into right and left pulmonary arteries, and after injections into either right or left pulmonary artery and pulmonary trunk.[10] Findings typical of atrial septal defect are shown in Figure 4. The curve from injection into the right pulmonary artery differs from that following injection into the left pulmonary artery in that the magnitude of the deflection is smaller and there is a well-marked second hump about six seconds after the first. These differences in deflections are due to a smaller amount of dye passing from the right lung into the left ventricle and systemic circulation. The remainder of the dye is shunted into the right atrium and passes again through the pulmonary circulation. The second peak is interval between the two peaks of concentration represents the average time which the dye takes to circulate once through the lungs. The finding of this difference between dilution curves from the right and left pulmonary artery demonstrates that in many cases of atrial septal defect a greater proportion of blood draining from the right lung is shunted in a left-to-right direction than is that from the left. This phenomenon probably is due to the proximity of the openings of the right pulmonary veins to the defect in the atrial septum.

In partial anomalous venous connection, the veins from one lung communicate directly with either the right atrium or one of its tributaries. This situation is associated with an anomalous drainage of the blood from that lung; hence, injections of dye into the right and left pulmonary arteries will serve to identify this condition in many instances. In the case of the anomalously draining right lung (Fig. 5) the appearance time is considerably prolonged, and the magnitude of the deflection is reduced in comparison with the curves from the normally draining left lung. If

equal volumes and equal rates of blood flow through each lung are assumed, then the prolonged appearance time following injection into the right pulmonary artery is due to the greater distance between the sites of injection and of sampling. This distance includes the pathway through the pulmonary vascular bed and right side of the heart which the dye has to traverse to reach the normally draining left lung which is the only route through which dye passes to the systemic circulation. Division of this dye in the pulmonary trunk into portions traversing the right and left lung reduces the total amount of dye passing to the systemic circulation and hence diminishes the magnitude of the resulting deflection. Recirculation of the remaining dye through the anomalous pathway causes a prolonged clearance of dye from the central circulation and, therefore, an increased disappearance time. When an anomalous pulmonary venous connection into the right atrium coexists with a defect of the atrial septum, it may not be possible to demonstrate the presence of an anomalous connection with certainty.*

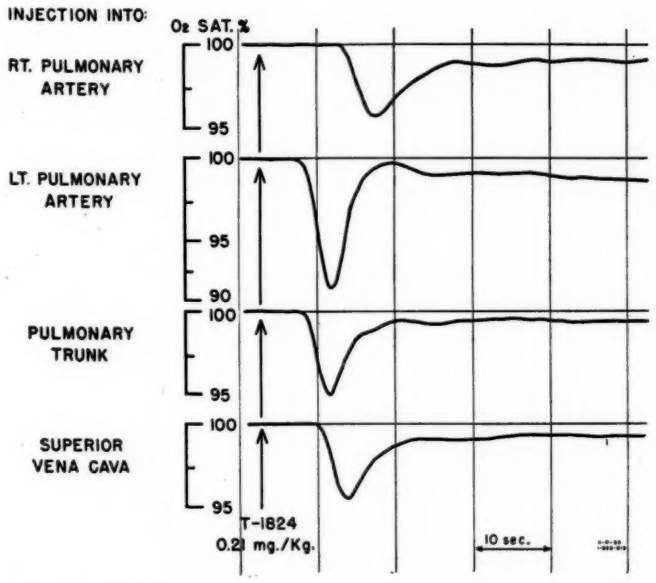


Fig. 5. Arterial dilution curves recorded following injection of T-1824 into right, left and main pulmonary arteries and superior vena cava of a forty-three-year-old woman with anomalous pulmonary venous connection and agenesis of the upper lobe of the right lung. The curve obtained after injection into the left pulmonary artery is normal (appearance time, 5 seconds). In contrast, curve obtained on injection of dye into the right pulmonary artery shows the appearance time is much prolonged (10.3 seconds), the magnitude of the deflection is reduced, and the disappearance slope is prolonged (for discussion see text).

Summary

Analysis of indicator-dilution curves based on differences in contour and time relationships offers a new tool in the study of heart disease. In particular the method permits a more detailed study of the dynamics in congenital heart disease and hence permits better appraisal of the anatomic defect present. In this presentation certain of the concepts basic to the technic have been outlined. In the individual case the physician at catheterization makes use of the methods as he proceeds with the study since careful selection of sites of injection on the basis of data being obtained during the procedure, will allow a more precise diagnosis to be made. In the cardiac catheterization laboratory of the Mayo Clinic this technic is an established integral part of the rou-

*This problem was discussed in some detail in a recent publication.[10]

tine investigation of the majority of cardiac patients studied. In these days of the increasing possibility of successful surgical therapy for congenital heart disease the added accuracy of diagnosis provided by the technic has proved to be of practical value.

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STROKE VOLUME CHANGES

(Continued from Page 115)

that of Dr. Remington in the assumption of a linear volume-pressure relationship, the slope of which is measured in each individual, while Dr. Remington accepts this individual variation by utilizing an average curve.

Since many assumptions and approximations are of necessity made in the derivation of the equations used, care must be taken to carefully control studies made with this technique by simultaneous determinations using independent methods. This is particularly true when used in conditions other than those already tested.

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MINNESOTA MATERNAL MORTALITY STUDY

The Maternal Mortality Committee of the Committee on Maternal Health of the Minnesota State Medical Association

THIS REPORT is the third concerned with the statewide maternal mortality study being carried out in Minnesota. The present report deals principally with the results of the current study, which includes the period April 1, 1951, through March 31, 1952. Certain comparisons are made between these results and those of the preceding year and with the results of a similar study carried out in Minnesota from July 1, 1941 to June 30, 1942. (The three surveys will hereafter be referred to as the "1951 survey," the "1950 survey," and the "1941 survey," respectively.)

The study of maternal deaths is sponsored by the Minnesota State Medical Association and the Minnesota Department of Health with the latter organization providing the necessary funds. Each death is thoroughly studied in a personal visit by a trained obstetrician, and the data so obtained is then considered in a completely impartial manner by the Maternal Mortality Committee of the Maternal Health Committee of the State Medical Association. The exact method by which the study is carried out has been detailed in a previous report.[1]

It is gratifying to note that a further reduction in the maternal mortality rate occurred during the period covered by the present report (Table I). For the purposes of this study, all deaths associated with pregnancy and those occurring during a period of three months postpartum are included, regardless of cause of death. In many instances it has been clear that the pregnancy or the postpartum state had no direct relationship to the cause of death, but these deaths are nevertheless included in this study for completeness. This group of cases constitutes the so-called non-obstetric deaths. The remaining deaths (excluding those due to chorionepithelioma which are also included in the present report) are those from which state and national maternal mortality rates are ordinarily calculated. Deaths due to chorionepithelioma are not included as maternal deaths in state and national rates, but are customarily included in reports on cancer. In Table I are shown the all-inclusive maternal death rates and the "maternal death" or corrected rates for each of the three years referred to. It can be seen that the reduction in the maternal death rate in Minnesota has been progressive and that the rate is consistently well below that of the United States generally.

TABLE I. MATERNAL DEATHS IN MINNESOTA
Studies for 1941, 1950, and 1951, Respectively

	Number Deaths	Number Births	Gross Mortality Rate Per 1000 Live Births†	Minn. Maternal Mortality Rate Per 1000 Live Births‡	U. S. Maternal Mortality Rate Per 1000 Live Births
1941	112	55,293	2.03	1.68	2.8*
1950	68	76,074	0.89	0.59	0.83**
1951	57	80,099	0.71	0.45	0.71**

*Average for 1941 and 1942.
**Unofficial.
†Including all maternal deaths from whatever cause.
‡Excluding deaths considered as non-obstetrical on the basis of the definitions set down by the United States Bureau of the Census. This is the ordinarily reported maternal mortality rate.

The comparison would be even more favorable to Minnesota were it not for the fact that in the three years of the study, thirty-nine cases have been added to the totals for this state by special efforts at case finding. These cases have included eight "obstetric deaths" and have been discovered principally by cross-matching birth and death certificates and by reports of various individuals interested in the study. In all thirty-nine instances, there was no mention of pregnancy or the postpartum state on the death certificate. Since the number of maternal deaths is customarily arrived at from information appearing on death certificates, none of the thirty-nine would have been included in the determination of maternal death rates had it not been for the special efforts undertaken to discover them. It is the opinion of the Committee that very few if any maternal deaths in Minnesota escape recognition under present circumstances.

Many factors are probably involved in the continuing reduction of maternal mortality. These include increased use of whole blood, the availability of various chemotherapeutic and antibiotic agents, and extensive use of hospitals for obstetrical deliveries. In the final analysis, the chief

TABLE II. PREVENTABILITY OF MATERNAL DEATHS IN MINNESOTA
Studies for 1941, 1950, and 1951, Respectively

	1941		1950		1951	
	No.	Per-cent	No.	Per-cent	No.	Per-cent
Preventable	82	73.2	22	32.4	24	42.1
Not preventable	27	24.1	45	66.1	32	56.1
Not determinable	3	2.7	1	1.5	1	1.8

TABLE III. CONSULTATIONS IN RELATION TO MATERNAL MORTALITY IN MINNESOTA
Studies for 1941, 1950, and 1951, Respectively

	1941		1950		1951	
	No.	Per-cent	No.	Per-cent	No.	Per-cent
None	63	56.2	29	42.6	28	49.1
Adequate	10	9.0	22	32.4	17	29.8
Inadequate	39	34.8	17	25.0	12	21.1

TABLE IV. AUTOPSIES IN RELATION TO MATERNAL MORTALITY IN MINNESOTA
Studies for 1941, 1950, and 1951, Respectively

	1941		1950		1951	
	No.	Per-cent	No.	Per-cent	No.	Per-cent
Obtained, adequate	9	8.0	24	35.3	23	40.4
Obtained, inadequate	27	24.1	2	3.0	3	5.2
Not requested	55	49.1	29	42.6	20	35.1
Permission refused	21	18.8	13	19.1	11	19.3

TABLE V. OBSTETRIC AND NON-OBSTETRIC DEATHS AMONG MATERNAL MORTALITY CASES IN MINNESOTA
Studies for 1941, 1950, and 1951, Respectively

	1941		1950		1951	
	No.	Per-cent	No.	Per-cent	No.	Per-cent
Obstetric deaths	93	83.0	46	67.6	39	68.4
Non-obstetric deaths	19	17.0	22	32.4	18	31.6

credit is obviously due to the practitioners in Minnesota who are engaged in practicing obstetrics and who are ultimately responsible for careful obstetric work and for applying to actual practice the advances being made. The achievement of a mortality rate that was considered to be the irreducible minimum at the time of the original state survey in 1941 must mean that the great majority of Minnesota physicians are administering extremely efficient obstetric care. The material collected in the three years of the survey shows real evidence of improvement in this care.

Perhaps the most noteworthy evidence of improvement concerns preventability of death, as shown in Table II. In 1941 almost three-fourths of the deaths were considered preventable, while in 1950 and 1951 only about one-third and two-fifths, respectively, were preventable. In each year the physician involved was charged with the responsibility for the great majority of the preventable deaths.

That some improvement has taken place with regard to consultations is indicated in Table III. It will be noted that adequate consultation was obtained in less than 10 per cent of cases in 1941, but that this percentage was more than tripled in 1950 and again in 1951. There is room for further improvement here. Consultation was thought to be indicated in an additional 25 per cent of cases in 1951.

The data in Table IV indicate a continuing increase in the relative numbers of autopsies being done. Since trained personnel are frequently not available for the performance of an autopsy, the figure of 40 per cent for adequate postmortem studies in 1951 is considered good.

Another indication of improvement in obstetric care is the increase in the percentage of deaths associated with pregnancy that are due to non-obstetric causes, as shown in Table V. In the last two years of the survey almost one-third of all the deaths were due to non-obstetric causes as compared with less than one-fifth of the 1941 deaths.

While the evidence of improvement in care is most encouraging, much more improvement is still possible as indicated by the material obtained in the studies. It is not assumed that the material covered by the study is at all representative of the practice of obstetrics in the state as a whole. Since all the patients died, it is much more likely that the material so gathered contains a considerable concentration of evidence of bad obstetrics. At the same time it does indicate where improvements can be made.

Adequate care as defined by the Committee in its list of minimum requirements is being given to a pitifully small percentage of the patients who die (Table VI).[1] That these deficiencies are not mere technicalities is indicated by the frequency with which various deficiencies in care contributed to the patient's death. The encouraging evidences of improvement cannot be allowed to obscure the fact that approximately

TABLE VI. MEDICAL CARE GIVEN IN MATERNAL MORTALITY CASES IN MINNESOTA
1951 Study

	Prenatal Care		Care in Labor and/or Delivery		Postpartum Care	
	No.	Percent	No.	Percent	No.	Percent
Adequate	6	13.6	11	25.5	14	42.4
Faulty, contributory	10	22.7	23	53.5	15	45.5
Faulty, not contributory	28	63.6	9	21.0	4	12.1
None	13		14		24	

TABLE VII. PELVIC MEASUREMENTS IN MATERNAL MORTALITY CASES IN MINNESOTA
Studies for 1941, 1950, and 1951, Respectively

	1941		1950		1951	
	No.	Percent of Registered Patients	No.	Percent of Registered Patients	No.	Percent of Registered Patients
Unregistered	18		6		13	
Not measured or incompletely measured	77	81.9	40	64.5	32	72.7
Measured	17	18.1	22	35.5	12	27.3

TABLE VIII. SEROLOGIC TESTS FOR SYPHILIS IN MATERNAL MORTALITY CASES IN MINNESOTA
Studies for 1941, 1950, and 1951, Respectively

	1941		1950		1951	
	No.	Percent of Registered Patients	No.	Percent of Registered Patients	No.	Percent of Registered Patients
Unregistered	18		6		13	
Not obtained	64	68.1	38	61.1	25	56.8
Obtained	30	31.9	24	38.9	19	43.2

TABLE IX. TYPE OF DELIVERY IN MATERNAL MORTALITY CASES IN MINNESOTA
Studies for 1941, 1950, and 1951, Respectively

	1941		1950		1951	
	No.	Percent of Registered Patients	No.	Percent of Registered Patients	No.	Percent of Registered Patients
Undelivered	23		17		21	
Spontaneous	39	43.8	24	46.2	16	44.5
Operative	50	56.2	28	53.8	20	55.5

40 per cent of the deaths occurring are still preventable. The number and variety of faults in management comprise a long list. In the majority of instances the disastrous outcome was not the result of lack of some specialized knowledge or ability on the part of the physician, nor to lack of facilities, but rather to failure to follow principles of care familiar to all physicians.

The following examples taken from the 1951 survey may serve to illustrate this point. One patient died of a ruptured uterus resulting from attempted podalic version and extraction following a prolonged obstructed labor at term. The obstruction was due to the presence of a large sarcomatous mass in the pelvis which was missed because a pelvic examination was not done. No fewer than seven patients were delivered by accouchement forcé, a procedure which is probably never indicated. Six of the seven died from hemorrhage as a direct result of the procedure. Three patients died as a result of complete lack of attention to fluid and electrolyte balance. Two of these were suffering from hyperemesis gravidarum while the third died on the eighth day after delivery by cesarean section. Despite the fact that the patient vomited frequently, had a negligible oral intake of food and fluid, and was subjected to gastric aspiration for more than three days, she was given no sodium, potassium or chloride until just before death and never more than 1,000 cc. of fluid parenterally daily.

Pelvic measurements are still obtained in only one-fourth of the cases (Table VII), and serology during the prenatal course in considerably less than one-half (Table VIII). Surely the value of these procedures does not need reemphasizing, yet the frequency with which they are done in the cases being studied is increasing only very slowly.

As indicated in Table IX, the relative incidence of operative deliveries is not changing significantly although the absolute incidence is. Approximately 40 per cent of the operative deliveries studied in the last two years of the survey were considered to be not indicated. More importantly, in 1951, 45 per cent were considered contributory to death.

Results of 1951 Survey

Table X contains the details regarding cause of death as determined by the Committee in the cases studied in 1951. Much of the information is self-explanatory but a few points require elaboration. It will be seen that hemorrhage was

TABLE X. PRIMARY CAUSES OF MATERNAL DEATHS IN MINNESOTA
1951 Study

Cause of Death		No.	Percent
Obstetric hemorrhage		13	22.8
Postpartum			
Lacerations of cervix and/or lower uterine segment with atony	9		
Intra partum	2		
Ruptured uterus	1		
Intra-abdominal hemorrhage (ectopic)	1		
Infection		6	10.5
Pulmonary embolism	4		
Septic abortion	1		
Generalized peritonitis of undetermined source	1		
Toxemia		4	7.0
Eclampsia superinposed on hypert. vasc. disease	3		
Eclampsia without pre-existing hypertension	1		
Chorionepithelioma		3	5.2
Anesthesia		3	5.2
Spinal	2		
Inhalation	1		
Dehydration and electrolyte imbalance		3	5.2
Hyperemesis gravidarum	2		
Post cesarean section	1		
Air embolism		2	3.5
Amniotic fluid embolism		1	1.9
Congenital heart disease with failure		1	1.9
Not determinable		3	5.2
Shock of unknown etiol. (? G.I. tract hem.)	1		
Sudden death late labor (? amn. fl. emb.)	1		
Threatened abortion—autopsy negative	1		
Non-obstetrical complications		18	31.6
Trauma	4		
Bulbar poliomyelitis	2		
Acute alcoholism	2		
Panmyelophthisis (chloramphenicol)	1		
Bilateral pneumothorax	1		
Probable rupture cerebral aneurysm	1		
Cerebral hemorrhage (hypersplenism)	1		
Pulmonary tuberculosis	1		
Malignant melanoma	1		
Carcinoma stomach	1		
Not determinable	3		
Totals		57	100.0

TABLE XI. ANALYSIS OF MATERNAL DEATH CERTIFICATES IN MINNESOTA
1951 Study

	No.	Percent
Complete and correct	18	31.6
Complete but incorrect	8	14.0
Incomplete and incorrect	13	22.8
Incomplete, otherwise correct	18	31.6
Totals	57	100.0

TABLE XII. ANALYSIS OF BIRTH AND STILLBIRTH CERTIFICATES IN MINNESOTA
1951 Study

	No.	Percent	Percent of Those Required
Complete and correct	18	31.6	48.6
Complete but incorrect	14	24.6	37.8
Incomplete, otherwise correct	5	8.8	13.5
Not required	20	35.1	
Totals	57	100.1	99.9

by far the leading cause of death. Almost one-half of the deaths due to hemorrhage were accounted for by the manual dilatations of the cervix previously referred to, none of which were indicated. Two of the remaining three patients who bled to death from lacerations of the birth canal had been given subcutaneous obstetrical pituitrin before delivery. One of the two cases of intrapartum hemorrhage involved a patient with a twin pregnancy who was sent back to bed after delivery of one twin. She died three and a half hours later of shock due to massive hemorrhage externally and into the uterus resulting from partial separation of the placenta. The second patient was delivered by an Indian midwife at home. The patient expired of hemorrhagic shock before the placenta was delivered.

The number of deaths due to infection continue to drop. As noted in Table X, pulmonary embolism accounted for two-thirds of the cases in this category.

A notable change between the 1950 and 1951 results concerns the toxemias. Toxemia was the leading cause of death in 1950, accounting for 19 per cent, whereas in 1951 the proportion had dropped to 7 per cent.

Failure to appreciate the intolerance of pregnant women to ordinary doses of anesthetic agents for spinal anesthesia accounted for two deaths. One patient received 150 mgm. of procaine and the other 18 mgm. of pontocaine. Both expired promptly of respiratory paralysis and shock.

Tables XI and XII are included to indicate the great need for improvement in the accuracy and completeness with which birth and death certificates are filled out. In eleven of the twenty-one incorrect death certificates, or 19.3 per cent of the total, the cause of death as determined by the Committee's investigation showed that the cause recorded on the death certificate by the physician concerned was incorrect.

Summary and Conclusions

1. A brief general summary of the results of the Minnesota Mortality Study for the period April 1, 1951, through March 31, 1952, has been presented. Certain comparisons are made between these results and those of the preceding year and with the results of a similar study carried out in Minnesota in 1941-1942.

2. There were fifty-seven deaths associated with pregnancy or the postpartum period in the 1951-1952 period among 80,099 live births for an over-all mortality rate of 0.712 per 1,000 live births. The rate when deaths due to non-obstetric complications are excluded was 0.45. This represents a further reduction as compared with the previous year's study and compares favorably with that of the whole United States. In 1951 the unofficial United States maternal mortality rate excluding non-obstetric complications was 0.71.

3. It is assumed that the remarkably low maternal mortality rate achieved means that efficient obstetric care is being administered by the vast majority of Minnesota physicians engaged in practicing obstetrics.

4. A significant reduction in the proportion of deaths that were considered preventable has taken place since 1941. Still more improvement is possible, since 42.1 per cent of the 1951 deaths were considered preventable.

5. There has been an increase in the percentage of deaths associated with pregnancy but due to non-obstetric causes—from 17 per cent in 1941 to 31.6 per cent in 1951. Thus, there is a significant decrease in deaths due to obstetric causes.

6. The percentage of adequate consultations obtained in 1951-1952 was more than three times that obtained in 1941.

7. Complete pelvic measurements were obtained in only 27.3 per cent of registered patients who died. This situation must be quickly improved. Serology was obtained in 43.2 per cent.

8. Of the delivered patients who died, 55.5 per cent were delivered by operative means. Of the operative deliveries, 40 per cent were considered not indicated and 45 per cent contributed to the patients' deaths.

9. As judged by the standard of minimum requirements for adequate obstetric care as adopted by the Maternal Mortality Committee, prenatal care was faulty in 86.3 per cent, care in labor and/or delivery was faulty in 74.5 per cent, and portpartum care was faulty in 57.6 per cent of the cases studied. The faulty care contributed to the patient's death in 22.7 per cent as regards prenatal care, 53.5 per cent as regards labor and delivery, and 45.5 per cent as regards postpartum care.

10. The primary cause of death is tabulated. Hemorrhage was the leading cause of death in 1951 with almost one-half of the deaths due to hemorrhage being accounted for by accouchment forcé. It is long past time for this procedure to be given up.

11. Adequate autopsies were performed in 40 per cent of the cases studied in 1951. This proportion is approximately five times the frequency with which autopsies were done in 1941.

12. Only 31.6 per cent of the death certificates and 48.6 per cent of the birth certificates reviewed were complete and correct. In 19.3 per cent, the cause of death as recorded on the death certificate was incorrect as determined by the Committee's investigation. The significance of this fact is clear. The validity of data obtained from unchecked death certificates is open at least to serious question.

13. The Committee would like to point again to the fact that in many of the hospitals in which these maternal mortalities are occurring, simple history taking, careful general and obstetric physical examination, and basic laboratory work are grossly deficient. That this is related to the end result for the patient is clear. The Committee would urge again that the medical and administrative staffs of such hospitals owe a duty to their patients to enforce the carrying out of these studies without exception. The minimum standards for adequate obstetric care as set down by the Committee are recommended.

14. The experience of the Maternal Mortality Committee would suggest that there are numerous sources of serious error in the usually reported maternal mortality rates. Case finding which depends on death certificate reports is incomplete and diagnoses from this source are grossly inaccurate. It is impossible to evaluate the accuracy of the division into so-called obstetric and non-obstetric deaths when only death certificate information is available.

15. The Committee is tempted to point out the justification for pride in the efficiency of obstetric care in Minnesota which these data indicate. It has overcome this temptation since there is clearly much to be improved and since pride and progress are uneasy bedfellows.

Reference

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INTERVERTEBRAL DISC LESIONS IN THE TEENAGE GROUP

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LOW BACK PAIN and mid-back pain with or without radiating pain into the leg is a fairly frequent complaint of both boys and girls in the teenage group. When a patient complains of lumbar or lumbosacral type pain one expects primarily to find evidence of some disease, such as tuberculosis, neoplasm or epiphysitis. When a patient in the teenage group, in addition to the back pain, complains of lower extremity radiation, one is highly suspicious of intervertebral disc type lesion. Radiating pain is not necessarily always present or associated with an intervertebral type lesion and in our series it is fairly common to have a well-established radiographic evidence of intervertebral disc pathology with no complaint of pain extending into either lower extremity. We have recently reviewed a rather large series of low back complaints in patients varying from sixteen to twenty years, nineteen of whom have come to laminectomy for an intervertebral disc lesion.

One of our greatest handicaps in the understanding and treatment of intervertebral disc lesion, both in teenagers and adults, is our limited knowledge of the cause of disc degeneration. There are many theories as to the cause of progressive degeneration in normal intervertebral discs, none of which have been definitely accepted by orthopedists. Because of the activity of both boys and girls in the teenage group it is quite common to attribute any progressive degenerative condition to repeated trauma or to one single episode of trauma. In spite of the fact that several of our intervertebral disc cases occurring in teenagers have been in association with athletic participation, we are rather impressed with the fact that the amount of precipitating trauma in this age group is rather slight.

All of us have seen a great many cases of low back pain in the teenage group which may have been associated with some minimal type lifting injury or athletic injury which would give rather marked difficulty for ten days or two weeks and then would spontaneously improve on bed rest and symptomatic treatment. A great many of these cases have a tendency to recur periodically but never reach the point of becoming bad enough to justify a spinogram or intensive work-up. I feel that in a great many of these cases there is progressive disc degeneration which perhaps represents the early phase in the large number of adult patients whose x-rays show long-standing degenerative disc changes. A very high percentage of these back complaints in teenagers respond very well to symptomatic treatment, including well-fitting wide lumbosacral belts. Certainly this age group, as well as adults should have a long period of conservative management before a laminectomy is considered. All of our patients who eventually require laminectomy were first given an extended period of initial bed rest, heat, sedation, and support.

I feel that in teenagers as well as adults with disc lesions and with unilateral sciatic radiation, only a small percentage actually obtain much improvement from traction. If traction is to be attempted it should only be for a trial period and if there is no definite improvement within forty-eight hours it should be discontinued. In the type of disc where a large free fragment is forced out of the intervertebral space and locked under the posterior ligament traction will definitely be of no value and will many times produce an increase of both back and leg pain. If, after the period of rest and conservative treatment there has been no improvement or insufficient improvement to allow return to activity further work-up is necessary. We feel that a spinogram is indicated in all these patients to more accurately localize the lesion and eliminate possibility of an unrecognized double disc.

I would like to briefly review nineteen cases of patients who eventually have come to laminectomy. Of these eight were girls and eleven were boys, the youngest of whom was sixteen and the eldest had presenting symptoms at the age of nineteen. Several writers have pointed out that in children and teenagers intervertebral disc lesions seem to be rather unusual in their lack of complaints of numbness, parasthesia, and muscle weakness. We have been unable to find any variation in presenting complaints or physical findings between the

teenage group and the adult group with similar lesions. Some writers have stressed the high incidence of lumbosacral disc lesions in the younger group but here again in our series eight cases had an L-4-5 defect; ten cases had an L-5-S-1 defect and one patient, a girl of seventeen, had at the same time a large defect at L-5-S-1 on the right and at L-4-L-5 on the left. The history of an injury period here again is quite variable. Only three had the beginning of back and leg complaints after athletic participation. Eight had a completely insidious onset of pain associated with no injury of any type. Two, a pharmacy student and a high school student, first had difficulty when bending over to tie their shoes. Four had difficulty after some minor lifting injury. One had an initial complaint of back and leg pain after sneezing and the last, the girl with the double-disc lesion, first had difficulty after falling a distance of approximately eighteen inches from a kitchen stool. On examination the physical findings in these patients were not unusual. The subjective complaints of shooting and radiating pain which was made worse by coughing, sneezing and straining, were the most common. In five patients out of the nineteen who had no shooting type, radiating pain, a herniated disc was suspected because of the rather long period of severe incapacitating back pain. All patients but three showed a very definite change in reflexes and five out of the nineteen had a definite change in sensation. None showed appreciable atrophy or definite muscle weakness.

Several authors have mentioned the finding of unusually stringy, or gelatinous, material obtained from the intervertebral disc space. This may be true in very young children with whom we have had no experience but in our cases the pathological specimens have been very little different from those in the adults. In five or six patients with large bulging discs which have been opened, a single fragment has been freely removable. The remaining material in the disc space has had a rubbery consistency and we have had the feeling that this represents an unripe or incompletely degenerated form of disc disease. It is in these patients that the prognosis is rather reserved. We make every effort to thoroughly curette the disc space and remove all such material but there is always the possibility of further separation of further degenerating disc material which may later give rise to a secondary protrusion from the same disc space. In the one patient of the nineteen who has had recurrent difficulty, a second laminectomy established the presence of more degenerated material at the same level and on the same side in rather large amounts. Except in these cases where an unripe disc is established at laminectomy we feel the prognosis in teenage patients is rather good. All patients are given a follow-up consisting of developmental back exercises and a gradual return to activity. Several of the patients have been able to return to rather strenuous athletic participation. One of our patients, against advice, was able to play first-string basketball at a Minnesota college three months after the laminectomy. Another patient has been able to satisfactorily play Big Ten football and has had no complaints after hard contact of line work.

Conclusion

In conclusion, I feel we should remember that true intervertebral disc lesions occur fairly frequently in the teenage group. Their treatment is chiefly conservative and only a small percentage require eventual laminectomy. The patients' presenting complaints and physical findings differ very little from adults. The prognosis in the patients not requiring laminectomy is rather good and in those requiring laminectomy the determining factor is the pathologic finding at operation. In cases in teenagers with complete separation of free fragment discs the prognosis is probably better than in adults with similar lesions.

THE FAMILY PHYSICIAN AND PUBLIC HEALTH

Health departments have traditionally worked with the full co-operation of the medical profession. The family physician is the front line of any public health endeavor. In order for a community health program to succeed, the practitioner must give full co-operation and active rather than passive support. Experience has shown that the more extensive the public health program a community enjoys, the greater the demand made by the public on services not only for the treatment of illness but also for health supervision and for preventive services.—Vlado A. Getting, M.D., J.A.M.A., Sept. 26, 1953.

Laboratory Aids to Medical Practice

Sponsored by
The Minnesota Society of Clinical Pathologists

RADIOACTIVE ISOTOPES IN DIAGNOSIS AND TREATMENT

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Section of Clinical Pathology
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NATURALLY occurring radioactive isotopes, such as radium and radon, have served medicine usefully for several decades. The discovery, in 1933, by Joliot and Curie that stable elements could be converted into artificially radioactive ones opened up undreamed possibilities that physicians are only beginning to realize.

The unstable (radioactive) isotopes or variants of naturally occurring elements achieve stability by means of reorganization of their nuclear structure. This reorganization is accompanied by emanations of various types, which include alpha, beta and gamma rays. As an example, one radioactive isotope of iodine (I^{131}) emits both beta and gamma rays; in so doing, its chemical properties are transmuted from those of iodine to those of xenon. The superscript number is used to identify the isotope, thus, I^{127} is the normal, stable form of iodine, while I^{131} is an isotope which weights 4 atomic units more. The various radioactive isotopes undergo nuclear reorganization at speeds varying from fractions of a second to billions of years. By convention these rates are expressed as the time required for the element to undergo half its radioactive degradation; thus, I^{131} has a "half life" of 8 days. In honor of Marie and Pierre Curie, codiscoverers of radium, the unit of radioactivity is the curie, which is approximately equal to the emanation from 1 gm. of radium. A thousandth and a millionth of a curie are designated as a "millicurie" (mc) and a "microcurie" (μc).

Radioactive isotopes are being used in medicine for therapeutic purposes and for tracer studies. The goal in treatment is to find an isotope with the following characteristics: (1) some degree of predilection for a certain organ or diseased cell, (2) sufficient strength of radiation to kill the diseased cells without serious injury to the patient as a whole; (3) no chemical toxicity and (4) a short half-life, so that the hazard of radiation is limited to days or at most weeks. To be able to concentrate a radioactive substance within a diseased portion of the body affords a means of intimate, continuous radiation without the interposition of normal tissues between the source and the target zone. Iodine, for example, tends to concentrate within the acinar epithelial cells of the thyroid gland; since these cells are unable to distinguish between stable I^{127} and radioactive I^{131}, administration of the latter leads to an internal source of radiation within hyperplastic or malignant thyroidal cells.

Radioactive colloidal gold (Au^{198}) settles out on pleural and peritoneal surfaces, affording a method of treatment of metastatic malignant lesions on these surfaces. Although cures by this method of treatment are rare, the relief of pain and diminution of effusion are often gratifying. Au^{198} may be injected directly into malignant tumors of the prostate, cervix or skin. Radioactive phosphorus (P^{32}), like its stable isotope, inclines toward regions of rapid growth, including bone marrow. This material lends itself readily, therefore, to treatment of the overactive erythropoiesis of polycythemia vera.

The other and perhaps more important medical application of radioisotopes is in the tracing of movements of elements within and between compartments of the body. The advantages of this method over direct chemical analyses are twofold. While one can measure chemically with precision the concentration of iodine (I^{127}) down to a few billionths of a gram per milliliter, one can detect radioiodine (I^{131}) in solutions still more dilute by a factor of several million. Secondly, if the radioactive element should emit gamma rays,

sensitive counters can detect the element from outside the intact body, because the gamma rays readily penetrate several inches of tissue and pass into the neighboring atmosphere. When a detector of radioactivity is placed over the thyroidal region of the neck and radioiodine is given intravenously, one can measure accurately the rate with which I^{131} concentrates in the thyroid, as well as the absolute amount extracted from the blood. Both these values are proving to be diagnostically useful in disease of the thyroid.

Many other diagnostic uses of radioisotopes, based on measurement of internal radioactivity from outside the body, are being investigated. These include: (1) localization of tumors of the brain, retina, breast and liver; (2) demonstration of block of the spinal canal; (3) evaluation of extravascular concentrations of blood; (4) estimation of the vascularity of skin grafts and (5) measurement of cardiac output and circulation time.

When samples of fluids or tissues can be removed from the body, more refined radioisotopic techniques become available. These methods often have an added meaning when chemical or biologic assay of the stable chemical is made along with determination of radioactivity. Globulins labeled radioactively are being studied by immunologists. Curiously enough, these studies are yielding results somewhat different from those obtained by labeling the same proteins with azo dyes. When serum albumin tagged with radioactive iodine is injected intravenously and allowed to mix thoroughly, the resulting calculated volumes for plasma and whole blood are as accurate as the results of dye methods and are simpler to obtain. Erythrocytes may be tagged with a variety of radioactive elements, which enables one to determine erythrocytic volumes and to estimate the longevity of these cells in various hemolytic states.

To the biochemist and the physiologist, radioactive substances are becoming indispensable accessories, particularly since the radioactive isotope of carbon (C^{14}) has become generally available. Even the bacteriologist has at his disposal bacteria in which radioactivity is incorporated from their nutrient medium.

Since the radioactivity of these various isotopes is potentially dangerous to both the patient and the investigator, only those familiar with the hazards are allowed by the Atomic Energy Commission, which is the sole agency authorizing their use, to handle radioisotopes. By virtue of his training, the radiologist is familiar with the benefits and dangers of radiation; he should be an integral part of the team to handle the isotopes. With expanding use of isotopes in diagnostic procedures, the clinical pathologist will find himself also a member of the team.

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This is the thirteenth in a series of editorial reports sponsored by the Minnesota Society of Clinical Pathologists and designed to foster closer relationships between the practicing physician and the pathologist.

The use of radioactive isotopes again emphasizes, as have several other of these reports, the ever-increasing role played by the laboratory physician in the modern practice of medicine. Although most physicians will not actively handle radioactive materials, they should be aware of the potential hazards associated with such factors as disposal of radioactive excreta from patients who have received radioactive isotopes.

Reprints of this report are available from the author or the chairman of the editorial committee.

GEORGE E. STILWELL, M.D.
Chairman, Editorial Committee

Mayo Clinic,
Rochester, Minnesota

RESIDENCY IN ANESTHESIOLOGY

The Army Medical Service residency program in anesthesiology will be increased in term to three years beginning July 1, 1954. to give candidates additional training, it has been announced by Major General George E. Armstrong, Surgeon General.

The new plan provides for the two years of clinical training required for American Board of Anesthesiology certification and an additional year devoted to research and development training co-ordinated with the basic sciences in anesthesiology.

According to the new plan, selected Medical Corps officers will spend their first year of the three-year residency at Walter Reed Army Medical Center, Washington, D. C., where they will receive fundamental clinical training.

The second year will be presented at the Army Medical Service Graduate School, Walter Reed Army Medical Center, Washington, and will be devoted to research and development.

The last year of residency will be spent at one of four Army hospitals and will present the candidates with a summation of training with clinical and teaching experience. The four hospitals to be utilized in the program are: Brooke Army Hospital, Brooke Army Medical Center, Fort Sam Houston, Texas; Fitzsimons Army Hospital, Denver, Colorado; Letterman Army Hospital, San Francisco, California; and Walter Reed Army Hospital, Walter Reed Army Medical Center, Washington, D. C.

President's Letter

DOCTOR AND PATIENT

The relationship of the doctor to his patient and the resulting public opinion of the profession as a whole should be uppermost in the minds of all doctors these days. In recent months, the entire medical profession has withstood many unpleasant attacks which have actually stemmed from the unethical practices of a small minority of practitioners.

Naturally, such articles bordering on the sensational, make excellent copy and rate high readership for both the author and the publications in which they appear. Medicine and medical news have reached a high point in lay readership, and as a consequence, any material of this nature is fair game for writers, and often results in unnecessary and unfair exploitation of the facts. It is unfortunate that only the substandard aspect of medicine should gain such notoriety.

On the official level, American medicine has reacted in various ways, ranging all the way from bitter indignation to complete disregard. Letters have been written to the editors, complete facts have been submitted, answering articles have been published, and verbal renunciations have been used.

However, would it be too much to ask of American medicine to revert back to the down-to-earth policy of improving its individual members' day-to-day relationships with the people who may hold some of those views—their patients? Physicians can do no better than that, to prove to the American people that they are conducting their practices on the highest possible level.

And, medicine is doing a job of policing itself on such matters. Medical organizations have established mediation boards with the express purpose of culling out those who infringe on medical ethics, and also to study any grievance against a member.

Another comeback to the sensational article writers has come to light recently. It is interesting and gratifying to see that some publications have printed articles pointing out this very fact—that the entire profession should not be condemned because of the unfortunate actions of a few.

With both writers and advertisers contributing, it is to be hoped that increased mutual good will and understanding will develop between doctors and patients.

Among the most recent items of this new pattern showing what American practitioners are really like are these: the January 11 issue of *Life* magazine carried an advertisement on "How to Select a Family Doctor;" the same ad appeared in the February 6 issue of the *Saturday Evening Post*, in February's *Woman's Home Companion*, February's *Parents Magazine* and in February's *Today's Health;* the November 1953 issue of *Better Homes and Gardens* carried an article entitled "When You Need a Doctor in a Hurry—What Will You Do?"; *Rotarian* of December, 1953 ran an article called "Don't Kill Your Doctor;" and *Look* of November 17, 1953 published a piece on how "Mississippi Trains Its Own Country Doctors."

These are only a few of the recent articles and advertisements appearing in national publications, which emphasize the positive side of American medicine. It is hoped that as a result of less sensational and more factual printed material, more and more people will realize that not all physicians adhere to the described unethical practices, and that the profession, as a whole, cannot be rightfully judged by the actions of the minority.

President, Minnesota State Medical Association

Editorial

ARTHUR H. WELLS, M.D., *Editor;* HENRY G. MOEHRING, M.D., and JOHN F. BRIGGS, M.D.

POLIO VACCINATION WITH A PRAYER

WE HAVE rejoiced with the recently announced prospects of a control for poliomyelitis. The series of discoveries from those of John F. Enders culminating in those of Jonas E. Salk reads like a streamlined version of Microbe Hunters. We delight in the thought that the National Foundation for Infantile Paralysis may have been instrumental in hastening these discoveries by many years. We want ever so much to have this scourge of mankind under control as soon as possible and at the same time see to it that those responsible get the proper recognition. However, as physicians, we soon learn after a continuous series of disillusionments to modify our immediate reaction "This is it!" to "This could be it."

In Minnesota, the State Board of Health, as the official public health agency of the state, is in the position where it must assume responsibility for the program of vaccination against poliomyelitis in 1954. The Board has given careful and continuing consideration to this matter since first approached. Several meetings have been held on the program with members of the State Polio Planning Committee; with the Council and other representatives of the Minnesota State Medical Association; with officials of the National Foundation for Infantile Paralysis; and consultation has been had by telephone and correspondence with scientists in other states, Canada, and Washington. The State Board, in addition, called together a special scientific advisory committee which met on January 20 to advise the board as to recommended procedures in the light of the latest available information. Developments since then have likewise been fully considered.

We wish to commend most highly the State Board for its cautious and yet complete approach to this important problem. The medical profession, as a group, has been constantly kept in the picture, and the advice of the best scientists in this field has been obtained. Everyone concerned has shown a desire to give wholehearted endorsement and support to the program of successful polio vaccination in Minnesota; yet throughout, care is being exercised to ensure complete safety of the procedure and the collection of sound data on which to evaluate the effectiveness of the vaccine. When the time comes for final plans, the medical profession will be glad to do its part.

ROUTINE CHEST ROENTGENOGRAMS IN PRIVATE HOSPITALS

IT is a matter of established fact that the most fertile field for the discovery of pulmonary lesions lies in the routine roentgen examination of the chests of patients being admitted to hospitals. It is also unquestionably a fact that hospitals should take all steps possible to protect their employes and patients from infectious tuberculosis. This is because of the responsibility, both moral and legal which the hospital must take. Therefore, it is of the utmost importance that hospitals make this relatively simple examination of all persons becoming associated with them, not only as a patients but also as employes or attendants in any capacity.

Most of the hospitals in Minnesota will be forced to make necessary adjustments for the examinations in their present accommodations. It is important that the examination unit be in as close proximity as possible to the admission area. The greater the distance between these departments the more difficult it is to obtain roentgenograms of patients, particularly of those admitted under trying circumstances, such as persons injured or those in labor. New hospital buildings being planned or constructed may easily make provisions for chest roentgenograms near or adjacent to the admission office. As a matter of fact, it seems that new hospital plants are now being arranged with the roentgen department on the entrance floor for greater convenience.

The type of routine roentgen examination does not have great significance. The method of making a roentgenogram in only one projection can be regarded as no more than a survey regardless of whether miniature or standard sized films are used. In general, it seems that hospitals of 150-bed capacity or less could best use 14 x 17-inch films made in the established roentgen department. On the other hand, larger institutions should be economically better off by investing in

a photoroentgen unit. These larger institutions would eventually repay the investment in a photoroentgen unit merely in the saving made on the examinations of new employes and the interval follow-up examinations of these employes.

The Minnesota Radiological Society has recommended that a charge of $1.50 be made to patients being admitted for photofluorograms and that a $4.00 charge be made for 14 x 17 films. These charges are in effect in those institutions employing either method in the Twin Cities. It is felt that these charges make the procedure self-supporting, which is no more than proper. A profit would probably defeat the purpose of the survey. On the other hand, the findings are of sufficient value to the individual being examined to make it worth his while to underwrite his share of the money and effort being expended by those responsible for the project.

It is apparent that processing of the films can best be done in the established roentgen department. It does not appear that there is any hospital in Minnesota sufficiently large to justify the establishment of a separate processing unit. It is cheaper and easier to provide the necessary material for developing and subsequent handling of film to last over a twenty-four-hour period than it is to construct and maintain separate facilities for this work. This also eliminates the need for a self-sustaining staff for the unit. Technicians, often students, can be employed to operate examination units in the larger institutions from 2 to 5 or 6 p.m. daily when most hospital admissions take place. During this time, these employes can also make roentgenograms of the chests of those persons admitted but not examined during the previous twenty-four hours. During the remainder of each day, the admitting office personnel can be made responsible for the procedure. It is a simple matter to train any reasonably intelligent person in the technical aspect of making a chest roentgenogram.

The keeping of records should be as uncomplicated as possible, as long as the records accomplish the primary purpose of the survey, namely—to protect patients and employes from infectious tuberculosis. The hospital *does* have the right to see to it that infectious tuberculosis be recognized or ruled out in any given chest lesion. Also the hospital has the right to insist that any necessary precautions be carried out when infectious tuberculosis is suspected. Beyond that, the individual members of the staff and medical organizations at the staff level and upward are responsible for subsequent diagnosis, observation and treatment of patient having lesions which are non-infectious.

Simplicity of record keeping saves time, money and personnel. It reduces the research value of the project. However, research should be left primarily to subsidized institutions. Hospitals depending for their support upon private fees are obligated to use those fees for private benefits. If the private hospital were to become engaged in a large scale follow-up program, elaborate records would become necessary. Keeping the fact in mind that the survey is made to discover infectious tuberculosis, a long-range follow-up program does not seem to be the responsibility of a private hospital. All positive findings should be reported to local or state health authorities for subsequent action. Such a plan could add greatly to the overall value of the projects under discussion.

It should be of interest to record briefly the most important findings in an admission survey made over a three-year period in one Minnesota private general hospital.* During the three years 17,790 patients had photofluorograms. The interpreter noted 2,307 positive findings. Twenty-six of these patients, previously unsuspected of having disease, are now undergoing institutional treatment for pulmonary tuberculosis. Three hundred sixteen are being observed or treated privately for suspected tuberculosis. Twenty-nine primary thoracic tumors were discovered, and twenty-one metastatic pulmonary lesions came to light. Eleven "coin" lesions were found, the majority of which are known to be static by reference to previous examinations or by subsequent examinations. Exact classification of these eleven lesions cannot be given because they have not been removed. Cardiac and aortic abnormalities accounted for 629 positive findings. Other findings, such as large calcified primary complexes, adhesions, anomalies, scoliosis, old fractures, et cetera, of doubtful or no clinical significance, accounted for most of the remaining positive reports. However, eighty pleural effusions (mostly congestive), two cases of silicosis, a pulmonary cyst and other interesting conditions were seen.

A routine admission film is unquestionably of great value in the study of general hospital patients. In addition to its primary purpose, it

*St. Joseph's Hospital, St. Paul, Minnesota.

frequently provides a short-cut to the final diagnosis and disposition of the patient. The total time of hospitalization is often reduced by a more speedy diagnosis. Discovery of pertinent findings not related to the reason for hospitalization may be made. We believe that routine chest examinations are more important in the detection of disease than routine urinalysis and blood studies.

J. P. MEDELMAN, M.D.

ROUTINE X-RAYING OF PATIENTS ADMITTED TO GENERAL HOSPITALS

THE x-raying of all admissions to general hospitals is an essential element of the tuberculosis control program. Most reports of hospital admission x-ray screening programs cite the discovery of unsuspected cases of disease, and relate an over-all percentage yield of tuberculosis that is higher than that found in the screening of general population groups. This is quite to be expected, for patients coming to hospitals are generally older people, and people with chronic diseases and vague symptoms. It is this group that usually participates poorly in general population surveys, which are directed at finding unsuspected disease in the apparently healthy person. Moreover, since some heart conditions and other chest disorders, as well as tuberculosis, can be detected by admission x-rays, good medical practice would seem to require that chest x-rays be taken on admission as routinely as blood tests and urinalyses.

The program of x-raying all hospital admissions represents an investment that pays multiple dividends to patients, hospital and community. Often, treatment for a previously undetected disease is received by the patient at an earlier date as a result of discovery by admission x-ray. In addition, the risk of infection within the hospital from an unsuspected case of tuberculosis is minimized for fellow patients, staff and personnel. And lastly, the chain of tuberculosis infection within the community is broken when a previously unsuspected case of tuberculosis is detected by the admission x-ray and brought under public health supervision.

Minnesota deserves congratulations on having one of the lowest tuberculosis death rates in the world in 1952. In fact, tuberculosis has not been among the ten leading causes of death in the state for the past three years. The achievement of this enviable record comes as no surprise, in view of Minnesota's vigorous execution of a progressive tuberculosis control program.

It is also gratifying to know that approximately forty of Minnesota's hospitals are routinely x-raying all admissions and that this program has the endorsement of the Minnesota State Medical Association, the Minnesota Department of Health, and the Minnesota Tuberculosis and Health Association. The fact that more than 2,000 previously unreported cases of tuberculosis are still being found each year in the state, emphasizes the necessity of intensifying case-finding efforts. Like the widely accepted techniques of surveying apparently healthy population groups, of x-raying residents and employes of mental and penal institutions, and of examining contacts and suspects, case-finding among hospital admissions is another extremely effective device which merits application. The general hospitals in Minnesota that do not routinely x-ray their patients on admission are urged to explore the possibilities of this important aspect of a tuberculosis control program.

Although x-raying all admissions presents the already heavily burdened general hospital with additional administrative and financial problems, many of these problems can be readily solved; the costs of the service are moderate, and the rewards are many. Coupled with recent improvements in treatment, the routine x-raying of some 13 million Americans entering general hospitals each year, would certainly bring us closer to the control of tuberculosis.

R. J. ANDERSON, M.D.

Doctor Anderson is Chief, Division of Chronic Disease and Tuberculosis, Public Health Service, U. S. Department of Health, Education, and Welfare.

MATERNAL MORTALITY SURVEYS

NATIONAL mortality rates were high and showed little change from 1915 to 1930. The average rate was 7.0 maternal deaths for every 1,000 live births. During this same period in Minnesota there was an average of 5.2 maternal deaths per 1,000 live births. Prior to the enactment of the Shepard-Towner Act of 1921 maternal health activities were carried on by a few state health departments or state medical associations. In 1917, the New York Academy of Medicine first became interested in the problem of puerperal mortality because death rates from puerperal causes had remained stationary while death rates from other preventable causes had been steadily declining. Our Division of

Child Hygiene, now known as the Section of Maternal and Child Health, was established by the Minnesota Department of Health in 1922. The Maternal Health Committee of the Minnesota State Medical Association was first appointed in 1936.

Because there was little change in the national maternal mortality rates for the previous ten years, the Obstetric Advisory Committee of the U. S. Children's Bureau, of which Dr. Fred L. Adair of Minnesota was a member and later the chairman of the American Committee on Maternal Welfare, in 1926 presented a plan of study of all puerperal deaths to be conducted in certain states in 1927 and 1928. Studies of maternal mortality based on death certificates were known to be insufficiently detailed to give an accurate picture of the situation. Accordingly, the study was to be carried out by means of personal interviews with the physicians attending all deaths from puerperal causes. The study was made in fifteen states, including Minnesota, at the formal request of each state medical society. The interviews were carried out by physicians on the staff of the State Boards of Health who visited the attending physician or midwife as well as the hospital concerned. The report was written under the guidance of the Obstetric Advisory Committee and was the first intensive and accurate study from which valid conclusions could be drawn. It furnished the impetus for subsequent studies which led to the establishment of maternal health committees in various parts of the country.

In 1930, the New York Academy of Medicine through its Committee on Public Health Relations began a detailed "study of the phases of the public health problems of obsetrics as they affect New York City." It was patterned along the lines of the survey made by Adair and his associates—the so-called "Fifteen States Study" and used the same questionnaire so that satisfactory comparisons would be possible. The three-year study was made in co-operation with the Department of Health of New York City and the Obstetrical Society of New York. The final conclusion of this study stated, "The hazards of childbirth in New York City are greater than they need be. Responsibility for reducing them rests with the medical profession." Coming from the medical profession itself and pointing out that two-thirds of deaths due to childbirth could be prevented if the care of the woman had been proper in all respects, the report aroused intense controversy and was followed by a storm of lay and medical criticism. Its publication, however, led to the formation of maternal mortality committees in many local groups and hospitals. A committee on maternal welfare was established by the Philadelphia Medical Society which is still actively functioning. Shortly afterwards such committees were organized by county medical societies of New York City, Cleveland, Boston and other areas.

This activity on the part of the medical profession in co-operation with public health authorities has been a major factor in the marked and continued decline in maternal mortality. Other factors that accelerated the rate of decline included better undergraduate training of physicians, increased postgraduate training and refresher courses in obstetrics, increased rate of hospitalization and more adequate hospital facilities and services, widespread use of chemotherapeutic agents and antibiotics, and increased availability and use of blood. In addition, public health measures such as the education of the public to the importance of early and adequate prenatal care as well as good obstetric care has played a significant role.

Information from the state health departments of all states and territories in 1951 indicated that maternal mortality surveys were being carried out in nineteen states and the District of Columbia. A number of larger cities also carry on similar studies, including Philadelphia, New York City, Syracuse, San Diego, and others. Twelve states send questionnaires to physicians and hospitals. A few limit their surveys to the information available on death certificates. Only eight states, including Minnesota, make a thorough investigation by the personal visit of trained obstetricians, considered by most authorities to be the only effective means of obtaining accurate and worthwhile data. Within the past year the Wisconsin State Medical Society in co-operation with the Wisconsin Department of Health has instituted a similar study. During the past two years the Minnesota Maternal Mortality Committee has received requests for detailed information on the operation and procedures of the study from eight state medical and obstetrical societies.

The first statewide maternal mortality study in Minnesota was carried out from July 1, 1941, through June 30, 1942, by the Maternal Mortality Committee of the Minnesota State Medical As-

(Continued on Page 160)

The Dean's Page

THE LYON LABORATORIES

On February 11, the University of Minnesota will dedicate the new medical building which is being named in honor of the late Elias Potter Lyon, Dean of the Medical School from 1913 to 1936.

This building, which joins Millard Hall and the Anatomy Building along Washington Avenue, contains four stories and a basement. The ground level is left open for a passageway into the court of the Medical School quadrangle.

The first floor of this building is divided into laboratories for histo-cyto-chemical research and is under the direction of Dr. David Glick, a national leader and authority in this field of work. The funds for this unit were provided by the National Heart Institute in order to increase facilities for research and for the training of personnel in this new and important field of micro-chemistry.

The second and third floors are assigned to cancer biology and provide facilities for the work of Dr. John J. Bittner and his associates, Dr. Carlos Martinez, Dr. Marthella Frantz, etc. One of these floors is devoted to laboratories, while the other houses thousands of pure strain inbred mice. Dr. Bittner is internationally known for his discovery of the "milk factor" in the development of hereditary mammary cancer in mice, as well as for other contributions to knowledge about genetic factors in cancers of mice. For this work Dr. Bittner recently received an award, the first made under a special bequest, from Middlesex College Hospital in London for the most important contribution to cancer research made anywhere in the world. The funds for the construction of these two floors were provided by the Minnesota Division of the American Cancer Society.

The fourth floor is devoted to bio-physics, a field of basic medical research of constantly increasing importance. Laboratories on this floor are utilized by Dr. Maurice Visscher and others in the Department of Physiology, and Dr. Karl Stenstrom and his colleagues in Radiation Therapy. The funds for this unit were provided by the National Cancer Institute.

The basement, as soon as funds for its completion can be obtained, will be devoted to air-conditioned animal quarters and associated research laboratories. This will enable us to use for other purposes the roof-house of Millard Hall which has been ill-suited and unsatisfactory for animal quarters.

Altogether this new building provides the Medical School with some long and urgently needed facilities for basic medical research. We invite the physicians of the state to visit this unit when they have an occasion or opportunity to come to the campus.

HAROLD S. DIEHL, M.D.,
Dean of the Medical Sciences

Medical Economics

Edited by the Committee on Medical Economics
of the
Minnesota State Medical Association
George Earl, M.D., Chairman

FTC FIGHTS DECEPTIVE ADVERTISING FOR HEALTH INSURANCE

A nationwide investigation into objectionable and deceptive advertising for hospitalization and medical care insurance has been initiated by the Federal Trade Commission. According to a recent issue of the Washington Report on the Medical Sciences, trade practice rules were promulgated by FTC in 1950, but this is the first large scale inquiry of its kind to be undertaken by the Commission.

Assistance of businessmen, state authorities and the public has been requested by Chairman Edward F. Howrey. Among the cases of alleged violations of trade rules already in FTC's hands are several which it received recently from William Langer, Republican senator from North Dakota, who is chairman of the Senate Judiciary Committee.

According to the Washington Report, "an estimated 800 health and accident companies, relying chiefly on advertising and mail-in coupons for their business, are in this highly competitive field today. There were fifty in 1925. In 1952, net premium payments totaled nearly $1.9 billion, which was an increase of 206 per cent over 1946."

CLAMOR FOR SECURITY REPORTED BY MRS. HOBBY

According to Mrs. Oveta Culp Hobby, secretary of the Department of Health, Education and Welfare, "the American people want more economic security."

Mrs. Hobby spoke to the third annual American Assembly recently, meeting in New York to discuss economic security problems. Mrs. Hobby pointed out that Federal Social Security was one of three foundations for the economic security of the individual, supplementing industrial pension and insurance plans and individual savings. She remarked that the present high rate of individual savings indicated that Social Security had not affected the incentive of individuals to provide for their own future, insofar as they are able.

According to a recent news issue from the Research Council for Economic Security, which reported the *New York Times'* account of the meeting:

> "It was stated that the conferees were in general agreement that the American people wanted more Social Security because 'they realized increasingly how little economic independence they had as individuals.' The consensus of the panels was that the average American had a feeling of insecurity and that such anxiety was justified by past experiences and the complexities of contemporary life. These factors, rather than a softening of national fibre, are responsible for concern over social security programs, the participants are reported to have found."

HOUSE, TOO, LOOKS AT SOCIAL SECURITY

Before the opening of the second session of the 83rd Congress in January, the fact-finding hearing on the Social Security program opened with much less flourish. The House ways and means subcommittee hearings were the preliminary to what promises to bring a very explosive battle over the whole philosophy of the old-age and survivors-insurance system. Evidences of this coming battle have already been seen in political events of the past month or so.

No matter how hot and heavy the battle, the committee, under Representative Carl T. Curtis of Nebraska, brought out some very useful and interesting information about Social Security costs and possible extension of coverage.

President Eisenhower has repeatedly asked for extension of Social Security to cover about 6,500,000 more persons, including self-employed farmers, domestic servants not now covered, and professional persons, with optional coverage to be

provided for an estimated 4,000,000 more, including clergymen and employes of state and local governments.

Present System Studied

The Committee spent considerable time studying the present system of Social Security, and brought to light some interesting facts and figures regarding the large amount of old-age-benefit dollars going abroad.

According to a recent issue of *Insurance Economics Surveys,*

"Records show that the $13,000,000 in Social Security benefits paid to beneficiaries abroad in 1952 exceeded the amount going to beneficiaries in 12 states here at home. These states were listed as Arizona, Delaware, Idaho, Montana, Nevada, New Mexico, North Dakota, South Dakota, Utah, Vermont, Wyoming, and Mississippi, mainly agricultural states where farmers are not covered by Social Security.

"It also brought out that such beneficiaries abroad are in a position to earn unlimited amounts on the side, whereas beneficiaries here forfeit their benefit payment if they earn more than $75 a month. Social security authorities admitted that they were stumped in finding a means of enforcing that stipulation abroad."

The American Medical Association has taken a stand opposing the inclusion of the medical profession in the extension of social security.

PRESIDENT PRESENTS HEALTH PROGRAM

President Eisenhower's State of the Union message to Congress brought forth many statements on general Administration policy on important medical matters. In summary, this is what he said:

Socialization of Medicine: "I am flatly opposed to the socialization of medicine. The great need . . . can best be met by the initiative of private plans. But it is unfortunately a fact that medical costs are rising and already impose severe hardships on many families. The federal government can do many things and still avoid the socialization of medicine."

Research: "The federal government should encourage medical research in its battle with such diseases as cancer and heart ailments, and should continue to help states in health and rehabilitation."

Hill-Burton Program: "The present . . . act should be broadened to assist in the development of adequate facilities for the chronically ill . . . of diagnostic centers, rehabilitation facilities and nursing homes."

Reinsurance of Health Plans: "The war on disease . . . needs a better working relationship between government and private initiative. . . . A limited government reinsurance service would permit the private and nonprofit insurance companies to offer broader protection to more of the many families which want and should have it."

Rehabilitation: "The program for rehabilitation of the disabled especially needs strengthening. . . . This program presently returns each year some 60,000 handicapped individuals to productive work. Far more disabled people can be saved each year . . . if this program is gradually increased."

Military Dependents: "Pay alone will not retain in the career service . . . the necessary numbers of long-term personnel. I strongly urge, therefore, a more generous use of other benefits important to service morale. Among these are more adequate living quarters, and medical care for dependents."

Medical Tax Deductions: ". . . we propose more liberal tax treatment for dependent children who work, for widows or widowers with dependent children, and for medical expenses."

Veterans: "The internal reorganization of the Veterans Administration is proceeding with my full approval. When completed, it will afford a single agency whose services, including medical facilities, will be better adapted to the needs of those 20 million veterans to whom the Nation owes so much." (There was no further reference to VA in the address.)

Social Security: "I ask that this extension (to 10,000,000 more persons) soon be accomplished. This and other major improvements . . . will bring substantial benefit increases and broaden the membership of the insurance system, thus diminishing the need for Federal grants-in-aid. . . ."

PLAN WOULD REINSURE VOLUNTARY HEALTH INSURANCE

The reinsurance of voluntary health insurance plans, referred to above, was asked of Congress by the Administration. The heart of the program probably would be a corporation set up with a federal appropriation for its original working capital. Once under way, its operations would be supported by payments from the member health insurance organizations of a percentage of their gross income from premiums. In some respects, it would be similar to the Federal Deposit Insurance Corporation for bank deposits.

According to a recent issue of the AMA Washington Letter, the reinsurance corporation device follows the general lines of the 1950 Wolverton bill. Mr. Wolverton was then a minority member of the House Interstate and Foreign Commerce Committee which must handle such legislation. Now he is chairman and in a position to effectively push his idea. The Eisenhower plan is under-

stood to include the following three major objectives of the Wolverton bill:

"1. Make it possible for health insurance plans to offer catastrophic or comprehensive coverage by having the federal corporation pay one-third or three-fourths of each survivor's claim in excess of $500 or $1,000.

"2. Make it possible for—or even require—the plans to offer coverage to individual subscribers.

"3. Require all participating plans to scale the cost of premiums in relation to family income. Families at the $5,000 or $6,000 level would pay the maximum, with those earning less paying less."

Other pertinent factors involved in this reinsurance plan are:

"Participating groups would pay about 2% of gross premiums collected to support federal corporation. . . . A limit would be placed on out-state subscribers. . . . Also a limit on over-the-schedule payments to physicians or hospitals. . . . Benefits would have to be clearly defined and meet standards set by the corporation. . . . Patients would have to be allowed free selection among participating hospitals and physicians. . . . Every licensed physician would be eligible to participate. . . . The Wolverton plan excludes commercial companies but the present administration program would admit them."

OREGON MEDICAL SOCIETY OPPOSES REINSURANCE

In line with the American Medical Association's opposition to the Administration plan for reinsurance of voluntary non-profit health insurance, the Oregon State Medical Society has already passed a resolution against such a proposition.

Quotes Supreme Court

Basing its opposition on the belief that reinsurance would actually subsidize the plans and therefore bring federal control, the Oregon organization states:

"The administration has declared that its proposed subsidies to voluntary health insurance plans do not involve 'government regimentation and control' in spite of the fact that the United States Supreme Court (1942) has declared that 'it is hardly lack of due process for the government to regulate that which it subsidizes.'"

The resolution further declares:

". . . the intervention of the federal government in the form of subsidies or otherwise in the operation of the voluntary health insurance plans is unwarranted in the face of the outstanding record of these plans which already have extended their benefits satisfactorily to increasingly large segments of our people, and are increasing the scope of their benefits as rapidly as is consistent with financial soundness. . . ."

Woman's Auxiliary

RAMSEY COUNTY REPORTS ON PROJECTS

The Women's Auxiliary to the Ramsey County Medical Society has completed several major projects since the beginning of activities for the year last fall.

The doctors' wives co-operated with the Fourth District Minnesota Nurses' Association in sponsoring panel discussions in various high schools throughout Saint Paul for nurse recruitment. Doctors' wives sat in on many of the panels, and aided in the transportation of speakers to and from the meetings. Mrs. Wallace Gleason served as chairman.

At the Board meeting in November, 1953, the Auxiliary made sixty cancer dressings.

The Ramsey auxiliary also co-operated in the annual Christmas Seal sale, helping to staff downtown booths with thirty-eight women. Total sales were $136.22. The auxiliary aided in the sponsorship of the annual tuberculosis essay contest among Saint Paul school children. Mrs. George Snyder served on the judging panel. The contest was won by Barbara Tauer of St. Joseph's school and by Ray Blacik of St. Agnes' school.

The January meeting was held at the Saint Paul College Club on January 25. Mrs. M. D. Hilker served as program chairman. The program emphasized civil defense and featured an excellent discussion by Mr. Donald Wood, executive secretary of the Association of Twin City Hospitals. Two films were shown: "One Survival" and "Emergency Action to Save Lives." At this meeting, the winners of the tuberculosis essay contest appeared to read their essays and also to receive $10 gift bonds from the Auxiliary.

AUXILIARY MEETS IN CROOKSTON

The Women's Auxiliary to the Red River Valley Medical Society met at the home of Mrs. D. E. Stewart, Crookston, Friday evening, January 8, 1954. The meeting followed a dinner at the Hotel Crookston with the Red River Valley Society. It also occurred on a night of wind and snow which forced many out-of-town visitors to remain in Crookston overnight.

Mrs. Stewart reported on subscriptions to *Today's Health*, and Mrs. J. F. Norman, of Crookston, gave the report on current legislation. Mrs. Norman is legislative chairman of the group.

Mrs. C. L. Oppegaard told about the regional meeting of the State Auxiliary organization at Detroit Lakes, and Mrs. A. R. Reff, of Crookston, reported on the orthopedic clinic held in Crookston in October.

Among out-of-town guests were Mrs. G. A. Sather, of Fosston; Mrs. C. L. Roholt, of McIntosh; Mrs. Clifford Stadem, of Twin Valley, and Mrs. B. R. Kinkade, of Ada.

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

230 Lowry Medical Arts Bldg., Saint Paul, Minnesota

E. M. Jones, M.D., Secretary

PHYSICIANS LICENSED FEBRUARY 13, 1953

January, 1953, Examination

Name	*School*		*Address*
ALARI, Heino	Eberhard-Karls U. Tubingen, Germany	MD 1947	Northwestern Hosp., Minneapolis, Minn.
ANDERSON, Hugh Vaux	U. of Oregon	MD 1952	3425 S.W. 9th, Portland, Ore.
BEDDOW, Ralph Marlowe	U. of Oregon	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
CRISP, Jr., Elmer Ernest	Northwestern U.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
DAVIS, Charles Truman	U. of Tennessee	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
DENKER, Arthur Gilman	U. of Oregon	MD 1952	63 E. Ash, Lebanon, Ore.
DUNN, William Joseph	U. of Illinois	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
ELSTNER, Howard L.	U. of Pittsburgh	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
ERICKSON, Robert Dell	U. of Minnesota	MD 1953	Wykoff, Minn
FISHER, Don Harvey	Northwestern U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
GALLO, Bela	Peter Pazmany U. Budapest, Hungary	MD 1945	Lake Bronson, Minn
GOSSELIN, Gilles	U. of Montreal	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
HARDY, Paul Edward	St. Louis U.	MD 1951	Ancker Hosp., St. Paul, Minn.
HEALY, John Russell	Jefferson Med. Col.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
JACKS, Quentin Douglas	U. of Mantitoba	MD 1940	102-110 2nd Ave. S.W., Rochester, Minn.
JARVIS, Bruce William	U. of Minnesota	MD 1953	1869 Marshall Ave., St. Paul, Minn.
LEPPO, Niilo Erkki Albert	U. of Helsinki Helsinki, Finland Candidate of Medicine	1931	503 Med. Arts Bldg., Duluth, Minn.
MacMILLAN, Angus Hugh	Licentiate of Medicine	1936	
McPHERSON, Alan Given	Queens U.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
	U.of Manitoba	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
MANSTEIN, George	U. of Pennsylvania	MD 1941	102-110 2nd Ave. S.W., Rochester, Minn.
MYERS, Richard Lyon	St. Louis U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
OLAVS, Olga	U. of Latvia Riga, Latvia	"Physician" 1942	1009 Nicollet Ave., Minneapolis, Minn.
PRICE, Mary Lomas	U. of Aberdeen Aberdeen, Scotland	MB SB 1947	102-110 2nd Ave. S.W., Rochester, Minn.
SATERSMOEN, Jr., Theodore	U. of Minnesota	MD 1948	Veterans Adm. Hosp., Dearborn, Mich.
SIBLEY, John Adams	U. of Chicago	MD 1946	102-110 2nd Ave. S.W., Rochester, Minn.
SKILLERN, Scott Dorsey	U. of Pennsylvania	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
STREITZ, John McDonald	U of Minnesota	MD 1952	Veterans Adm. Hosp., Minneapolis, Minn.
VAN PROOYEN, Cornelia Mary	Washington U., Mo.	MD 1951	Mpls. Gen. Hosp., Minneapolis, Minn.

Reciprocity Candidates

Name	*School*		*Address*
BEST, Etta Wright	U. of Louisville	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
CARROLL, John Joseph	Creighton U.	MD 1947	117½ S. 1st St., Montevideo, Minn.
COOK, Clayton Stevens	Tulane U.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
FELDAKER, Mauri	Washington U., Mo.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
GOFF, John Robert	U. of Rochester	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
GUNN-SMITH, Allan Murray	U. of Minnesota	MD 1950	431 10th Ave., Two Harbors, Minn.
HEIST, Erwin William	U. of Iowa	MD 1951	Jaguum Hosp., Lutheran Mission, Madang, Terr., New Guinea
HUGHES, Sidney Osborne	U. of Pennsylvania	MD 1947	172 Main St., Winona, Minn.
HYMAN, Jr., Orren Williams	U. of Tennessee	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
JESSE, Richard Henry	U. of Nebraska	MD 1949	Truman, Minn.
KEYS, John R.	U. of Cincinnati	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
KUNTZ, Aloysius Leo	Tulane U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
LEMMON, Mark Leonard	U. of Texas	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
O'LEARY, James Michael	Jefferson Med. Col.	MD 1946	102-110 2nd Ave. S.W., Rochester, Minn.
OPFELL, Richard William	U. of Iowa	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
PERRY, Jr., John Francis	U. of Texas	MD 1947	U. of Minn. Hosp., Minneapolis, Minn.
SANDERS, Edward Joseph	Creighton U.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
SLEDGE, Claire Blount	U. of Oklahoma	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
SPITTEL, Jr., John Allen	U. of Maryland	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.

Name	*School*		*Address*
TOON, Robert Wallace	U. of Oregon	MD 1944	1251 Med. Arts Bldg., Minneapolis, Minn.
TROUP, Richard Henry	Northwestern U.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.

National Board Candidates

Name	*School*		*Address*
ALLEN, George Seaver	U. of Rochester	MD 1950	Mineral Spgs. San., Cannon Falls, Minn.
BAGLEY, Russell Willis	Tufts Med. College	MD 1948	871 S. Wilder St., St. Paul, Minn.
BEEMAN, Edward Arthur	Boston U.	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
BENJAMIN, Robert Bedell	U. of Minnesota	MD 1952	18th & Taylor, Hood River, Ore.
BOUCHELLE, McLemore	Harvard U.	MD 1943	6th Ave. & 9th St. N., Virginia, Minn.
BUCKLEY, Joseph John	New York Med. Col.	MD 1946	500 Union St., Minneapolis, Minn.
CHAPIN, Lemuel Edward	Temple U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
GORDON, Donald Lloyd	New York Med. Col.	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
LIPINSKI, Stanley Wnuk	Northwestern U.	MD 1951	Hibbing, Minn.
SILVER, Lawrence	New York Univ.	MD 1950	Hosp. of Rockefeller Inst. for Med. Research, 66th St. & N. Y. Ave., New York 21, N. Y.
SVIGALS, Robert Elliott	New York Med. Col.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
VAN BUSKIRK, Charles	Albany Med. Col.	MD 1947	U. of Minn. Hosp., Dept. Neurology, Minneapolis, Minn.
WEBB, Jr., Wilson Davenport	New York Med. Col.	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
YOUNG, John Van Winkle	Northwestern U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.

PHYSICIANS LICENSED MAY 22, 1953

April, 1953, Examination

Name	*School*		*Address*
AMBRUS, Laszlo	Eberhard-Karls U. Tubingen, Germany	MD 1948	462 S. Robert St., St. Paul, Minn.
AMBRUS, Milka	Eberhard-Karls U. Tubingen, Germany	MD 1948	Midway Hosp., St. Paul, Minn.
ANDERSON, Jack G.	Marquette U.	MD 1951	Mpls. Gen. Hosp., Minneapolis, Minn.
BALEISIS, Peter	U. of Dusseldorf	MD 1949	700 W. Broadway, Minneapolis, Minn.
BARLOW, Loren Call	Northwestern U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
BOZANIC, Milos S.	Ludwig Maximilian U. Munich, Germany "Arzt"	1948	St. Joseph's Hosp., St. Paul, Minn.
CONNAUGHTON, Patrick Alex	St. Louis U., Mo.	MD 1951	Mpls. Gen. Hosp., Minneapolis, Minn.
DATESMAN, Robert Wallace	U. of Pennsylvania	MD 1951	USAH, 3444 ASU, Camp Stewart, Ga.
DE WEESE, Jr., Robert Cornell	U. of Colorado	MD 1952	St. Cloud Nat. Bk. Bldg., St. Cloud, Minn.
FEINBERG, Milton	U. of Illinois	MD 1941	2215 Plymouth Ave. N., Mpls., Minn.
FOLEY, III, Philip James	Northwestern U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
GLENNY, Jr., William Robb	U. of Minnesota	MD 1953	Box 308, Osseo, Minn.
GREENWOLD, Warren Eldon	U of Chicago	MD 1946	102-110 2nd Ave. S.W., Rochester, Minn.
HEPBURN, Allan Lockwood	U. of Alberta	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
JOHNSON, Marvin Waldemar	U. of Minnesota	MD 1953	Milaca, Minn.
KARLINSKY, William	U. of Manitoba	MD 1945	644 Niagara St., Winnipeg, Man., Can.
KARNOPP, Katherine	U. of Oregon	MD 1952	Glen Lake San., Oak Terrace, Minn.
KONIG, Theodore John	U. of Minnesota	MD 1952	Veterans Adm. Hosp., Minneapolis, Minn.
LASZEWSKI, Franz von Zelberschwecht	Westphalen Wilhelms U. Munster, Germany	MD 1929	St. Joseph's Hospital, St. Paul, Minn.
LYSYJ, Anatol	Ludwig Maximilian U. Munich, Germany	MD 1950	624 W. 54th St. S., Minneapolis, Minn.
McCULLOCH, Charles Fraser	Queens Univ.	MD 1945	102-110 2nd Ave. S.W., Rochester, Minn.
MORISSETTE, Gaston	Laval Univ.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
PLACE, Virgil Alan	Johns Hopkins U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
PROSHEK, Lumir Charles	U. of Iowa	MD 1949	Mpls. Gen. Hosp., Minneapolis, Minn.
RICHARDS, Albert MacDonell	U. of Minnesota	MD 1948	Heart Hosp., U. of Minn., Mpls., Minn.
RYZEN, Nicholas	U. of Tennessee	MD 1951	Anoka State Hosp., Anoka, Minn.
SABANAS, Alvina Olga	U. Vytautas the Great Kaunas, Lithuania	MD 1939	102-110 2nd Ave. S.W., Rochester, Minn.
SMITH, Dale Compton	U. of Kansas	MD 1945	102-110 2nd Ave. S.W., Rochester, Minn.
SOUIRES, Franklin John	U. of Manitoba	MD 1945	102-110 2nd Ave. S.W., Rochester, Minn.
THOMPSON, Richard Eugene	Northwestern U.	MD 1951	NPBA Hosp., Glendive, Mont.
TORKELSON, Leonard Beck	U. of Minnesota	MD 1953	St. Joseph's Hosp., St. Paul, Minn.
TSAI, Shih Hao	National Med. Col. Shanghai, China	MD 1941	Glen Lake San., Oak Terrace, Minn.
VOIGHT, Allan Earl	U. of Oregon	MD 1951	Mpls. Gen. Hospital, Minneapolis, Minn.
VREELAND, Oliver Henry	U. of Oregon	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
WESTPHAL, Paul Harry	U. of Minnesota	MD 1953	Gettysburg, S. D.
WOYDA, William Charles	U. of Minnesota	MD 1953	Box 609, Elk River, Minn.

Reciprocity Candidates

Name	*School*		*Address*
BOSSARD, John Wesley	U. of Maryland	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
BOYD, George Knox	U. of Pittsburgh	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
BURGERMAN, Arthur	Indiana U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
BUTLER, Jr., Lewell Colbert	Louisiana State U.	MD 1946	102-110 2nd Ave. S.W., Rochester, Minn.
CAYCE, John Hill	U. of Texas	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
CLINGER, Edwin James	Ohio State U.	MD 1946	102-110 2nd Ave. S.W., Rochester, Minn.
COOPER, Milton	Cornell U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
DURAN, Robert Jackson	U. of Oklahoma	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
FYKE, Jr., Frazier Earl	U. of Pennsylvania	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
HUEBERT, Dan W.	U. of Kansas	MD 1946	26 Washington, Hutchinson, Minn.
JACKSON, Robert Carl	U. of Wisconsin	MD 1945	102-110 2nd Ave. S.W., Rochester, Minn.
JOHNSON, Robert Hugo	U. of Nebraska	MD 1952	210 3rd St. S.W., Little Falls, Minn.
KILMER, Warren Lee	Indiana U.	MD 1952	State Bk. Bldg., Inter. Falls, Minn.
LAW, William McConnell	Med. Col. of Va.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
LEE, Philip Randolph	Stanford U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
LOCHRIDGE, Jr., Edwin Payne	U. of Georgia	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
LOWE, James Cecil	U. of Tennessee	MD 1943	102-110 2nd Ave. S.W., Rochester, Minn.
MacLEAN, Donald Beacom	Wayne Univ.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
PRICE, Jr., William Edmund	U. of Oklahoma	MD 1946	412 Delaware St. S.E., Minneapolis, Minn.
PURO, Henry Evert	Standford U.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
QUINONES, Rafael Enrique	U. of Tennessee	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
REDELFS, John Wright	U. of Nebraska	MD 1945	102-110 2nd Ave. S.W., Rochester, Minn.
RICKMAN, James H.	U. of Louisville	MD 1946	102-110 2nd Ave. S.W., Rochester, Minn.
RYAN, Robert Frank	Stanford U.	MD 1947	102-110 2nd Ave. S.W. Rochester, Minn.
SCHWARTZ, Carl Alfred	U. of Pennsylvania	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
SCOTT, Jr., Charles	Emory Univ.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
SMITH, Jr. Fred Newman	Tulane Univ.	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
TAYLOR, Harry Edwin	Temple Univ.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.

National Board Candidates

Name	*School*		*Address*
EDELMANN, Robert Benedict	Long Island Col. Med.	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
GARAMELLA, Joseph John	Georgetown U.	MD 1945	Veterans Adm. Hosp., Minneapolis, Minn.
HARTRIDGE, Virginia Bishop	Woman's Med. Col. of Pa.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
O'NEIL, Richard Lawson	Boston Univ.	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
WALDMANN, Edward Bernard	Creighton Univ.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
WANNAMAKER, Lewis William	Duke Univ.	MD 1946	Dept. Ped., U. of Minn., Hosp., Minneapolis, Minn.
WINN, Jr., William Edward	Harvard Univ.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.

PHYSICIANS LICENSED JULY 17, 1953

June, 1953, Examination

Name	*School*		*Address*
ANDERSON, Richard William	U. of Minnesota	MD 1953	St. Joseph's Hosp., St. Paul, Minn.
ARNESON, Genevieve A.	U. of Minnesota	MD 1953	U.S.P.H.S. Hosp., New Orleans, La.
BAKKE, Arnold Clifford	U. of Minnesota	MD 1953	St. Joseph's Hosp., St. Paul, Minn.
BASINGER, Harold Preston	U. of Minnesota	MD 1953	Williamsport Hosp., Williamsport, Pa.
BEARMAN, Morton	U. of Minnesota	MD 1953	Ancker Hosp., St. Paul, Minn.
BERG, Arnold Martin	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
BOWLIN, Paul Frederick	U. of Minnesota	MD 1953	Oakland Naval Hosp., Oakland, Calif.
BRETZKE, Carl Otto	U. of Minnesota	MD 1953	Greenbush, Minn.
CAMMOCK, Earl Elarth	U. of Minnesota	MD 1953	Harborview Hosp., Seattle, Wash.
CARR, William Johnson	U. of Minnesota	MD 1953	338 LaSalle Bldg., Minneapolis, Minn.
COUNTRYMAN, Ethlyn	U. of Minnesota	MD 1953	San Francisco Hosp., San Francisco, Cal.
CROUCH, Boyden L.	U. of Kansas	MD 1946	102-110 2nd Ave. S.W., Rochester, Minn.
DEMARAIS, Francis Emery	U. of Minnesota	MD 1952	2508 Colfax Ave. S., Minneapolis, Minn.
DOBSON, Mervin Wilbert	U. of Manitoba	MD 1944	Medical Center, Mankato, Minn.
DOXSEE, George Charles	U. of Iowa	MD 1952	30 N.W. 1st St., Chisholm, Minn.
DUNN, Robert Clarence	U. of Minnesota	MD 1953	St. Joseph's Hosp., St. Paul, Minn.
FILIPOVICH, Orest Nicholas	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
FRENCH, Bayard Taylor	U. of Iowa	MD 1952	2229 3rd Ave. E., Hibbing, Minn.
FUSARO, Ramon Michael	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
GODFREY, H. Wilson	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
GOODCHILD, William Russell	U. of Minnesota	MD 1953	Mt. Sinai Hosp., Minneapolis, Minn.
GRANQUIST, Richard David	U. of Minnesota	MD 1953	St. Luke's Hosp., Duluth, Minn.
HABERLAND, Lyle Freeman	U. of Minnesota	MD 1953	Sacramento Co. Hosp., Sacramento, Cal.
HAGEDORN, Jr., Max Boykin	Loyola U.	MD 1949	Veterans Adm. Hosp., Minneapolis, Minn.
HALVERSON, William Gearey	U. of Minnesota	MD 1953	St. Mary's Hosp., Duluth, Minn.

Name	*School*		*Address*
HANSON, Daniel James	U. of Minnesota	MD 1953	Mercy Hosp., Toledo, Ohio
HANSON, Harold Walden	U. of Minnesota	MD 1953	St. Joseph's Hosp., St. Paul, Minn.
HEID, James K.	U. of Minnesota	MD 1953	Madigan Army Gen. Hosp., Fort Lewis, Wash.
HEINZERLING, Carl Robert	U. of Illinois	MD 1953	Mt. Sinai Hosp., Minneapolis, Minn.
HOPPERSTAD, Jule Jerome	U. of Minnesota	MD 1953	St. Mary's Hosp., Minneapolis, Minn.
HOYT, Charles Sherman	U. of Minnesota	MD 1953	Valley Forge Army Hosp., Phoenixville, Pa.
HUFF, John Stephen	U. of Minnesota	MD 1953	Memorial Hosp., Phoenix, Ariz.
JOHNSON, Gordon Erik	U. of Minnesota	MD 1953	St. Luke's Hosp., Duluth, Minn.
JUDE, James Roderick	U. of Minnesota	MD 1953	Johns Hopkins Hosp., Baltimore, Md.
KADESKY, Harold Baird	U. of Minnesota	MD 1953	2300 W. 50th St., Minneapolis, Minn.
KALLENBACH, Rudolf Werner	U. of Minnesota	MD 1953	703 Armstrong Blvd. S., St. James, Minn.
KANE, John Francis	Marquette U.	MD 1952	117½ S. 1st St., Montevideo, Minn.
KAPLAN, Martin Jack	U. of Minnesota	MD 1953	Michael Reese Hosp., Chicago, Ill.
KARON, Everett Howard	U. of Minnesota	MD 1953	U. of Minn. Hosp., Minneapolis, Minn.
KATKOV, Harold	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
KENT, James Roy	U. of Minnesota	MD 1953	St. Luke's Hosp., Duluth, Minn.
KETOLA, Tauno Everett	U. of Minnesota	MD 1953	Highland Alameda Co. Hosp., Oakland, Cal.
KIRSCHBAUM, Thomas Harry	U. of Minnesota	MD 1953	U. of Minn. Hosp., Minneapolis, Minn.
LARSON, Earl Russell	U. of Minnesota	MD 1953	King Co. Hosp., Seattle, Wash.
LENTON, John David	Creighton U.	MD 1953	Creighton Memorial-St. Joseph's Hosp., Omaha, Neb.
LEUZINGER, Donn Ellis	U. of Minnesota	MD 1953	U.S.P.H.S. Hosp., Seattle, Wash.
LINDALL, Dale Regnar	U. of Minnesota	MD 1953	Tripler Army Hosp., Honclulu, Hawaii
LINNELL, Leonard S.	U. of Minnesota	MD 1953	St. Luke's Hosp., Duluth, Minn.
LITIN, Robert Beryl	U. of Minnesota	MD 1953	Santa Clara Co. Hosp., San Jose, Cal.
LITTLE, Bruce Raymond	U. of Minnesota	MD 1953	Santa Monica Hosp., Santa Monica, Cal.
MAGUIRE, Gerald Eugene	U. of Minnesota	MD 1953	Sacramento Co. Hosp., Sacramento, Cal.
MARK, Aaron Louis	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
MARTIN, Jr., George Burnie	U. of Illinois	MD 1950	117½ S. 1st St., Montevideo, Minn.
McFARLANE, Donald Ray	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
McMAHAN, Robert Oliver	U. of Minnesota	MD 1953	U. S. Naval Hosp., Oakland, Cal.
MEAGHER, Thomas Arthur	U. of Minnesota	MD 1953	King Co. Hosp. System, Seattle, Wash.
MELBY, James Christian	U. of Minnesota	MD 1953	U. of Minn. Hosp., Minneapolis, Minn.
MELLER, Maurycy	U. of Parma, Italy	MD 1937	First Nat. Bk. Bldg., Brainerd, Minn.
MILLER, Harold Dawes	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
MURN, Thomas Gregory	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
NACHTIGAL, Fred C.	U. of Minnesota	MD 1953	Veterans Adm. Hosp., Long Beach, Cal.
NAHAS, Gabriel	U. of Toulouse, France	MD 1944	315 Millard Hall, U. of Minn., Minneapolis, Minn.
NASH, Eldore Bentley	U. of Minnesota	MD 1953	Memorial Hosp., Phoenix, Ariz.
NEILSON, William George	U. of Illinois	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
NORQUIST, Joseph Louis	U. of Minnesota	MD 1953	Walter Reed Army Hosp., Washington, D. C.
ODLAND, Donald Mark	Marquette U.	MD 1943	Mpls. Gen. Hosp., Minneapolis, Minn.
OLSON, Gregory Mathias	U. of Minnesota	MD 1953	Sacramento Co. Hosp., Sacramento, Cal.
PARSONS, Roger Alan	U. of Minnesota	MD 1953	St. Joseph's Hosp., St. Paul, Minn.
PERKINS, Douglass Everal	U. of Minnesota	MD 1953	614 Broadway, Alexandria, Minn.
POPADIUK, Peter	Friedrich Alex. U. Erlangen, Germany	MD 1948	St. Mary's Hosp., Minneapolis, Minn.
PRESTON, Frank Stebbins	U. of Minnesota	MD 1953	St. Vincent's Hosp., New York, N. Y.
PUGSLEY, Robert Daniel	U. of Minnesota	MD 1953	Swedish Hosp., Seattle, Wash.
RAMLOW, Ralph Marlyn	U. of Minnesota	MD 1953	Ancker Hosp., St. Paul, Minn.
RICHMAN, Harold Gene	U. of Minnesota	MD 1953	Barnes Hosp., St. Louis, Mo.
RICHTER, David James	U. of Illinois	MD 1951	6th Ave & 9th St. N., Virginia, Minn.
ROCKWELL, Winthrop R.	U. of Minnesota	MD 1953	Tripler Army Hosp., Honolulu, Hawaii
RUDIE, William Donald	U. of Minnesota	MD 1953	St. Mary's Hosp., Duluth, Minn.
SCHAFER, Thomas Leo	U. of Minnesota	MD 1953	Detroit Receiving Hosp., Detroit, Mich.
SCHMIDT, Jr., Erwin Rudolph	U. of Wisconsin	MD 1952	St. Luke's Hosp., Duluth, Minn.
SCHNEIDER, James A.	U. of Minnesota	MD 1953	Gorgas Hosp., Panama, Canal Zone
SCHREINER, Leon H.	Northwestern U.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
SETZER, Jr., Hobert Joseph	U. of Minnesota	MD 1953	Mpls. Gen. Hosp., Minneapolis, Minn.
SHIELDS, Jack William	Stanford U.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
SISLER, David Allen	U. of Minnesota	MD 1953	Grand Rapids, Minn.
SONTAG, David W.	U. of Minnesota	MD 1953	Veterans Adm. Hosp., Long Beach, Cal.
SUNBERG, Ruth Dorothy	U. of Minnesota	MD 1953	U. of Minn. Hosp., Minneapolis, Minn.
TAYLOR, Robert Owen	U. of Minnesota	MD 1953	San Diego Co. Gen. Hosp., San Diego, Cal.
TEYNOR, Joseph William	U. of Minnesota	MD 1953	U. S. Naval Hosp., Great Lakes, Ill.
THAL, Sam Harold	U. of Washington	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
TIFFANY, Francis Buchanan	U. of Minnesota	MD 1953	St. Luke's Hosp., New York, N. Y.
ULRICH, Christian Andrew	Northwestern U.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
VERMUND, Halvor	U. of Oslo, Norway	MD 1943	U. of Minn. Hosp., Minneapolis, Minn.
VORIS, David Clarence	Toulouse, France		102-110 2nd Ave. S.W., Rochester, Minn.

WALDRON, John Fossen	U. of Minnesota	MD	1953	Mpls. Gen. Hosp., Minneapolis, Minn.
WALTER, William Edward	U. of Minnesota	MD	1953	Mpls. Gen. Hosp., Minneapolis, Minn.
WATERS, Alvin W.	U. of Minnesota	MD	1953	Ancker Hosp., St. Paul, Minn.
WATSON, William Richard	U. of Minnesota	MD	1953	Fitzsimons Army Hosp., Denver, Colo.
WEGNER, Marwood Emery	U. of Minnesota	MD	1953	U.S.P.H.S. Hosp., Staten Island, N. Y.
WENZEL, James Brandrup	U. of Minnesota	MD	1953	Memorial Hosp., Phoenix, Ariz.
WILLIAMS, Paul A.	U. of Minnesota	MD	1953	Tripler Army Hosp., Honolulu, Hawaii
WILLIAMS, Richard Albert	U. of Iowa	MD	1952	Rothsay, Minn.
ZELLMER, Richard Edward	U. of Minnesota	MD	1953	U.S.P.H.S. Hosp., Baltimore, Md.
ZEMMERS, Roberts	U. of Latvia Riga, Latvia	"Physician"	1926	St. Luke's Hospital, Duluth, Minn.

Reciprocity Candidates

Name	*School*			*Address*
BEITHON, Paul Jule	U. of Nebraska	MD	1952	Miller Hosp., St. Paul, Minn.
BURLEIGH, Jr., Edward Gilbert	Louisiana U.	MD	1952	More Hosp., Eveleth, Minn.
CALVERT, Wilson Clark	Northwestern U.	MD	1951	102-110 2nd Ave. S.W., Rochester, Minn.
COLLINS, Jr., Robert Aaron	U. of Georgia	MD	1948	102-110 2nd Ave. S.W., Rochester, Minn.
DYER, John Allen	U. of Pennsylvania	MD	1947	102-110 2nd Ave. S.W., Rochester, Minn.
JOHNSON, Morris Blaine	U. of Nebraska	MD	1952	Miller Hosp., St. Paul, Minn.
LEITSCHUH, Robert Bernard	Marquette U.	MD	1946	Sleepy Eye, Minn.
LEWIS, Jr., Royce Clay	Tulane U.	MD	1946	102-110 2nd Ave. S.W., Rochester, Minn.
McCALL, J. Preston	Baylor U.	MD	1948	102-110 2nd Ave. S.W., Rochester, Minn.
McKAY, Walter Edward	Indiana U.	MD	1948	102-110 2nd Ave. S.W., Rochester, Minn.
MOBLEY, Jack Ervin	Vanderbilt U.	MD	1948	102-110 2nd Ave. S.W., Rochester, Minn.
RACE, George Alexander	Temple U.	MD	1943	102-110 2nd Ave. S.W., Rochester, Minn.
SILAS, Ralph Maurice	U. of Michigan	MD	1943	Veterans Adm. Bldg., No. 18, Ft. Snelling, St. Paul, Minn.
SIMPSON, Joseph Ralph	Louisiana State U.	MD	1947	102-110 2nd Ave. S.W., Rochester, Minn.
SMITH, Jr., Ross Haugh	U. of Pittsburgh	MD	1951	102-110 2nd Ave. S.W., Rochester, Minn.
SOMMERNESS, M. Duane	Temple U.	MD	1945	Willmar State Hosp., Willmar, Minn.
THATCHER, Wilbur Clayton	U. of Iowa	MD	1933	Physicians Bldg., Fort Dodge, Ia.
WALLACE, III, Alexander	Baylor U.	MD	1948	102-110 2nd Ave. S.W., Rochester, Minn.
WILLER, Stanley Hermann	Ohio State U.	MD	1948	102-110 2nd Ave. S.W., Rochester, Minn.

National Board Candidates

Name	*School*			*Address*
ALEXANDER, Jr., John Dewey	U. of Pennsylvania	MD	1949	102-110 2nd Ave. S.W., Rochester, Minn.
ALTHAUSEN, Jr., Theodore L.	U. of California	MD	1947	65 S. 10th St., Minneapolis, Minn.
BRIDGE, Allyn Gustave	Yale U.	MD	1948	301 Kenwood Pkwy., Minneapolis, Minn.
DE WALL, Richard Allison	U. of Minnesota	MD	1953	1833 2nd Ave., Anoka, Minn.
FARLEY, Frank Giles	Syracuse U.	MD	1945	2229 3rd Ave. E., Hibbing, Minn.
GROCH, Sigmund Noel	Tufts Col. Med. Sch.	MD	1949	102-110 2nd Ave. S.W., Rochester, Minn.
LINDNER, Janus Casimir	Yale U.	MD	1950	505 Nicollet Ave., Minneapolis, Minn.
MARR, Jr., Norval Mason	Cornell U.	MD	1948	102-110 2nd Ave. S.W., Rochester, Minn.
NICHOLSON, Richard Waldo	Harvard U.	MD	1948	Heron Lake, Minn.
RUTLEDGE, John Boyden	U. of Minnesota	MD	1952	1050 Lake Ave., Detroit Lakes, Minn.
SKRDLA, Willard Blake	U. of Nebraska	MD	1948	102-110 2nd Ave. S.W., Rochester, Minn.

PHYSICIANS LICENSED NOVEMBER 13, 1953

October, 1953, Examination

Name	*School*			*Address*
ALLBAUGH, Robert Dean	Washington U., Mo.	MD	1952	Mpls. Gen. Hosp., Minneapolis, Minn.
AMICK, Lawrence Douglas	U. of Iowa	MD	1945	Sac City, Iowa
BONNET, John David	Johns Hopkins U.	MD	1952	102-110 2nd Ave. S.W., Rochester, Minn.
BOTHAM, Richard James	U. of Wisconsin	MD	1952	102-110 2nd Ave. S.W., Rochester, Minn.
BRUNSTING, Jr., Louis Albert	U. of Michigan	MD	1952	102-110 2nd Ave. S.W., Rochester, Minn.
CAMPAGNA, Mario Joseph	U. of Oregon	MD	1952	102-110 2nd Ave. S.W., Rochester, Minn.
CARPER, John Mark	Jefferson Med. Col.	MD	1952	102-110 2nd Ave. S.W., Rochester, Minn.
CHRISTU, Chris Nicholas	Louisiana State U.	MD	1952	102-110 2nd Ave. S.W., Rochester, Minn.
COMESS, Morton Seymour	U. of Illinois	MD	1952	102-110 2nd Ave. S.W., Rochester, Minn.
CUMMISKEY, Paul Vernon	U. of Minnesota	MD	1953	104 S. 4th St., Mankato, Minn.
DEVANNEY, Louis Raynor	Northwestern U.	MD	1939	102-110 2nd Ave. S.W., Rochester, Minn.
DOAN, Robert English	U. of Minnesota	MD	1953	Mpls. Gen. Hosp., Minneapolis, Minn.
DOUGLASS, Jesse Ellsworth	U. of Minnesota	MD	1952	2325 Stinson Blvd., Minneapolis, Minn.
HENDRICKSON, Russell Robert	U. of Minnesota	MD	1953	Cook Co. Hosp., Chicago, Ill.
HIGGINS, John Alden	U. of Minnesota	MD	1953	102-110 2nd Ave. S.W., Rochester, Minn.
HOWARD, David Lloyd George	U. of Toronto	MD	1948	102-110 2nd Ave. S.W., Rochester, Minn.
HUNTER, Samuel Wynne	U. of Rochester	MD	1947	U. of Minn. Hosp., Minneapolis, Minn.
JOHNSON, David Arthur	U. of Minnesota	MD	1953	Mt. Sinai Hosp., Minneapolis, Minn.
LOUGHEED, Lawrence Edward	U. of Manitoba	MD	1951	102-110 2nd Ave. S.W., Rochester, Minn.

Name	*School*		*Address*
MAHLE, James Parker	U. of Minnesota	MD 1953	St. Joseph's Hosp., St. Paul, Minn.
McSWEENY, II, Austin J.	Loyola U.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
MORROW, Jr., George William	U. of Illinois	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
MOULTON, Keith Buell	U. of Minnesota	MD 1953	St. Joseph's Hosp., St. Paul, Minn.
NORDLUND, Mildred Eva	U. of Chicago—Rush	MD 1929	Cass Lake, Minn.
NORMAN, Jr., Mark Lewis	Marquette U.	MD 1952	1764 Grand Ave., St. Paul, Minn.
OLWIN, Thomas Kent	U. of Oregon	MD 1952	Ancker Hosp., St. Paul, Minn.
OZOLINS, Marta Birzins	U. of Latvia Riga, Latvia	MD 1927	274 Iglehart Ave., St. Paul, Minn.
PEAKE, Ronald Alan	Indiana U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
PITTELKOW, Robert Bernard	Marquette U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
POPOVICH, Dragojla, nee'Ostojich	U. of Belgrade Belgrade, Yugoslavia	MD 1939	Northwestern Hosp., Minneapolis, Minn.
RAILE, Richard Bayles	U. of Utah	MD 1950	1354 E. Maynard Dr., St. Paul, Minn.
RENO, III, George Louis	St. Louis U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
SAFIRESCU, Sorin Radu	U. of Bologna, Italy	MD 1950	Fairview Hosp., Minneapolis, Minn.
SEYBOLD, Herbert McCelvey	U. of Texas	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
STEWART, Allan H.	Geo. Wash. U., D.C.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
SVARE, Gerhart Trygve	U. of Wash., Seattle	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
TAINTOR, Ronald W.	U. of Louisville	MD 1952	Ancker Hosp., St. Paul, Minn.
TERRY, Howard Lincoln	Northwestern U.	MD 1944	Anoka State Hosp., Anoka, Minn.
THOMPSON, Murray Cornwall	U. of Western Ont.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
TRAYNOR, Jr., Mack Vincent	Northwestern U.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
VANDEVER, Harry Wallace	U. of Tennessee	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.
VEINBERGS, Arnolds	U. of Latvia "Physician" Riga, Latvia	1940	Swedish Hosp., Minneapolis, Minn.
ZIMERING, Sabina Szwarc	Ludwig Maximilian U. Munich, Germany	MD 1950	2741 Chicago Ave., Minneapolis, Minn.

Reciprocity Candidates

Name	*School*		*Address*
BETTS, Charles Samuel	Vanderbilt U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
BLATTSPIELER, Lucien H.	U. of Nebraska	MD 1946	Worthington, Minn.
CANTRELL, Glenn	U. of So. Calif.	MD-1947	102-110 2nd Ave. S.W., Rochester, Minn.
CHRISTIANSON, Herbert B.	Marquette U.	MD 1939	102-110 2nd Ave. S.W., Rochester, Minn.
CONGDON, Gordon Hall	U. of Chicago	MD 1940	102-110 2nd Ave. S.W., Rochester, Minn.
CREAMER, Dick Oliver	Baylor U.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
DeLAND, II, Frank Howard	U. of Louisville	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
ELLIOTT, Dean Cook	Ohio State U.	MD 1946	102-110 2nd Ave. S.W., Rochester, Minn.
ELLWOOD, Jr., Paul Murdock	Stanford U.	MD 1953	U. of Minn. Hosp., Minneapolis, Minn.
FAUCETT, Robert Lynn	U. of Kansas	MD 1944	102-110 2nd Ave. S.W., Rochester, Minn.
FONTANA, Robert Scott	St. Louis U.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
FOX, LeRoy John	Northwestern U.	MD 1945	NPBA Hosp., St. Paul, Minn.
HELMHOLZ, Jr., Henry F.	Johns Hopkins U.	MD 1937	102-110 2nd Ave. S.W.. Rochester, Minn.
HONATH, Donald Herbert	Wayne U.	MD 1947	111 W. Main St., Owatonna, Minn.
KIRK, Jr., Thomas Allen	U. of Virginia	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
LEAVENS, Milam Edmund	Baylor U.	MD 1948	102-110 2nd Ave. S.W.. Rochester, Minn.
LIMBECK, Donald Arthur	U. of Nebraska	MD 1950	State Hosp., Fergus Falls, Minn.
LOES, Louis Augustine	U. of Iowa	MD 1947	1225 St. Germain St., St. Cloud, Minn.
MILLER, John William	U. of Louisville	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
MORGAN, Edward Raymond	Tulane U.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
MUDD, Robert Hiden	U. of Pennsylvania	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
PIFER, Lawrence Harrison	U. of Indiana	MD 1952	1218 N. 3rd St., St. Joseph, Mo.
REIFSNYDER, III, Wm. Henry	Jefferson Med. Col.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
RITTER, Donald Glenn	Indiana U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
TERRY, Jr., Howard Richards	State U. of N. Y.	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
TURNER, Jr., John Cooper	Tulane U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
WALKER, Jr., Harry Charles	Med. Col. of Va.	MD 1951	U. of M. Grad. Sch., Minneapolis, Minn.
WALKER, Jr., Jack Andrew	Southwestern Med. Col.	MD 1948	102-110 2nd Ave. S.W., Rochester, Minn.

National Board Candidates

Name	*School*		*Address*
BIRKHEAD, Newton Charles	U. of Pennsylvania	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
BOTTI, John Dominic	U. of Pennsylvania	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
BUIE, Jr., Louis Arthur	U. of Minnesota	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
DOYLE, Jr., John B.	Geo. Wash. U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
ENSRUD, Earl Richard	Northwestern U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
FABI, Mario Nestor	Geo. Wash. U.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
FOX, Irwin J.	New York Med. Col.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
GLASS, Bernard	Ohio State U.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.

MINNESOTA STATE BOARD OF MEDICAL EXAMINERS

Name	*School*		*Address*
GREENFIELD, Duane Leonard	U. of Maryland	MD 1946	833 S. Phillips Ave., Sioux Falls, S. D.
HADER, Marvin	New York Med. Col.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
HELME, William Beach	Loyola U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
KOGL, Richard Conrad	U. of Minnesota	MD 1953	Veterans Adm. Hosp., Minneapolis, Minn.
LEIGH, James Arthur	Bowman Gray Sch. of Med.	MD 1952	606 N. 3rd St., East Grand Forks, Minn.
MAGNESS, John Lower	Geo. Wash. U., D. C.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
MISRAHY, George Albert	Harvard U.	MD 1948	State Hospital, Hastings, Minn.
O'HOLLERAN, Lloyd Stephen	Creighton U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
PEMBERTON, Henry Walter	Geo. Wash. U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
PETERSEN, Deane Alfred	U. of Nebraska	MD 1946	1409 Willow St., Minneapolis, Minn.
RANDALL, Alan Douglas	Tufts Col. Sch. Med.	MD 1950	102-110 2nd Ave. S.W., Rochester, Minn.
ROTHWELL, Walter Spray	Harvard U.	MD 1947	102-110 2nd Ave. S.W., Rochester, Minn.
SHRINER, John Franklin	Tulane U.	MD 1950	1826 Clearmont St., Mobile, Ala.
STEINMETZ, Rodney Dunlap	Northwestern U.	MD 1952	102-110 2nd Ave. S.W., Rochester, Minn.
TWEEDDALE, Duane Newton	Creighton U.	MD 1951	102-110 2nd Ave. S.W., Rochester, Minn.
UPDIKE, II, Edwin Hoyt	Columbia U.	MD 1949	102-110 2nd Ave. S.W., Rochester, Minn.
WEISBART, Myron Herbert	Columbia U.	MD 1950	Veterans Adm. Hosp., Minneapolis, Minn.
WILLIAMS, Winfield Tyler	U. of Minnesota	MD 1952	Veterans Adm. Hosp., Minneapolis, Minn.

MEDICAL STUDENT ENROLLMENTS

Says the *Journal of the American Medical Association*† disclosing the 53rd annual report on medical education of its Council on Medical Education and Hospitals, "enrollments in the country's 72 medical and seven basic science schools during 1952-53 totaled 27,688, or 2.3 per cent more than the 27,076 enrolled during 1951-52. The 5,668 students graduated during the last year exceed by 279, or 4.4 per cent, the previous record established in 1947, when at the termination of the wartime accelerated program several schools graduated more than one class. The estimated number of graduates for 1953-54, based on enrollments reported for senior classes in schools, is even greater—6,831." There was, however, a slight decrease in the size of the entering freshman class for the first time in five years—7,425, or 16 less than the record class of 1952-53.

In addition to those regularly enrolled as full-time students of medicine, medical schools had enrolled a total of 140 part-time and special students working toward an M.D. degree, and 407 students in internships that were a part of the degree requirements of the medical schools at which they were educated. There was also a total of 1,734 students from the United States enrolled in 72 foreign medical schools, located in 22 different countries.

There were 1,463 women in medical and basic science schools during 1952-53, eight less than last year, the report said. The women comprised 5.3 per cent of all students, as compared with 5.4 per cent last year. Women in freshmen classes totaled 399, against 394 last year. They constituted 6.1 per cent of all applicants to medical schools this year, compared with 5.6 per cent last year. The actual number of women graduates increased slightly —363, or 5.5 per cent of the total, as contrasted to 351 last year. The record number of women graduates—612, was set in 1949.

A total of 715 Negro students, 2.6 per cent of all enrollments, was reported.

The number of veterans enrolled in medical and basic science schools declined for the second year. There were 7,942 veterans enrolled, comprising 28.7 per cent of the total student body, as compared with 11,436, or 42.2 per cent, last year. There were 36 women veterans.

For the fourth consecutive year, the number of applicants for admission to medical schools continued to decrease, the report disclosed. During the year 1952-53, 16,763 individuals applied for admission, a decrease of 3,157 from 1951-52, and 5,516 less than the number who applied in 1950-51.

"This report has emphasized in recent years that the difficulty of gaining admission to medical schools has been grossly exaggerated," it was stressed.

"The statistics presented here amply confirm this exaggeration. The situation in the past year, when there were 2.6 applicants for each place in medical schools, was approximately comparable to the year 1929-30 when there were 2.6 applicants for each available place.

"One major factor is the disappearance of the large backlog of veterans who completed their premedical training following the termination of World War II. Another factor, one that is certain to cause serious concern among medical educators, is the sharp decrease in the number of applicants with superior academic records.

"While, again, one can only speculate as to the cause, it seems likely that this drop is in no small measure due to increasing competition for such talent from other professional fields."

Medical faculties and medical students alike show a continuing interest in education for family or general practice, the report pointed out. A high proportion of the medical schools sponsor one or more programs specifically designed to induce students to this type of practice.

The report also disclosed that physicians of this country are cognizant of the fact that medical education is a continuous process and that it is their responsibility to keep abreast of advances in the field of medicine. During 1952-53, 64,608 physicians attended 1,341 postgraduate courses.—Editorial, *New York State Journal of Medicine*, February 1, 1954.

†J.A.M.A., 153:105 (Sept. 12) 1953.

American Medical Association

Proceedings of the House of Delegates

Clinical Meeting

St. Louis, Missouri, December 1 to 4, 1953

The Clinical Meeting of the American Medical Association's House of Delegates, December 1 to 4, 1953, in St. Louis, Missouri, was notable for an absence of the controversies and stormy debate that have characterized some of the meetings of recent years.

The delegates were chiefly concerned with the application, readjustment and implementation of programs and policies already initiated. They embarked upon few new undertakings, but to policies already adopted they gave strong support, and dissenting voices were few.

Several restatements of national policies were made for purposes of clarification. Among them were the Association's policies with respect to medical care for dependents of military personnel, also medical aspects of any program for universal military training which in the future may be passed by Congress. A new emphasis was likewise placed upon the established policy for extension of voluntary prepayment plans for medical and hospital service and the work of the Council on Medical Education and Hospitals was buttressed, in spite of some expressed dissatisfaction, in its expanded accreditation program and its participation in the so-called "Matching Intern Program."

A resolution clarifying membership status and a number of other progressive changes in the Constitution and By-Laws will be welcomed by the membership. Also the adoption of definite objectives for the new Committee on Alcoholism, initiated last year as part of the Council on Mental Health, and of a medical association policy for participating in a national blood bank.

As a result of the end of hostilities in Korea, the doctor-draft law ceased to be a major issue at this session, but the delegates approved the suggestion that Congress be asked to eliminate the draft of doctors entirely at its forthcoming session.

Dependent Policy Restated

So long as a defense army is maintained, however, the question of medical care for dependents of military personnel is likely to be an issue. Restatement of the policy laid down at the interim session in Denver was especially pointed up by consideration of the report of the Citizen's Advisory Commission on Medical Care of Dependents of Military Personnel, the so-called Moulton Commission, which favors continuing and even expanding such care by military establishments. The statement adopted by the House of Delegates declared that the recommendations of the Moulton Commission "would completely reverse the generally accepted position that civilian facilities, if available and adequate, should be utilized first and that the use of military facilities should be restricted to emergencies and to dependents overseas and in remote areas." The statement will now be sent to the Secretary of Defense, to the chairmen of Senate and House military affairs committees and to the Commission on Re-organization of the Executive Branch of the Government, the Hoover Commission. The fact that President Eisenhower in his State of the Union address to Congress subsequently asked for increased medical service for dependents of military personnel makes it certain that this issue will assume especial importance in the months to come.

The policy adopted with respect to any future program of universal military training which may be passed by Congress is in line with the stand on dependent care and with the vigorous opposition voiced at the June meeting against medical service for veterans with non-service-connected disabilities. All have as their objective the placing of definite limitations on the amount of medical service to be provided by the federal government which may otherwise soon expand to a point where the private system of medical service and the quality of care available to the public in general is seriously threatened.

Medical Students Should be Excluded

If any plans are being laid now for early adoption of universal military training, medical students should be excluded from consideration until after they are graduated from medical school, the delegates believe. Their policy also calls for pre-induction examinations and periodic check-ups during the reserve period to be performed by civilian physicians in order to avoid serious withdrawals from hard-pressed civilian medical services. It calls for rehabilitation and corrective treatment which might be found necessary in pre-induction examinations to be carried out, likewise, by civilian doctors in civilian institutions and it asks that trainees in any contemplated reserve corps be ruled ineligible for veterans' medical benefits unless their disabilities are actually connected with active military service.

The delegates proposed that a civilian board be set up nationally to insure a proper balance between civilian and military needs. In that con-

nection, also, they approved an official request from the association that a physician be retained under the reorganization plan in the position of Assistant Secretary of Defense (health and medical). The position is held at the present time by Dr. Melvin A. Casberg.

In order to carry forward the campaign initiated by the American Medical Association last June against federal medical care for veterans with non-service-connected disabilities, with stipulated exceptions, each constituent association was urged by the delegates to create a special committee on veterans' medical service. Several regional conferences on the subject have already been held and they will be extended to all parts of the country to acquaint doctors with AMA policy, it was announced.

Membership Clarified

Many delegates were happy to have the somewhat confused membership picture in the American Medical Association cleared up, as far as delinquents are concerned. They approved the following resolution as a substitution for a resolution introduced by Dr. George Earl of the Minnesota delegation last June. The latter had proposed that delinquent AMA dues be cancelled for members of any constituent state association which should subsequently make AMA membership compulsory for all its members. The substitute resolution adopted at St. Louis reads as follows:

RESOLVED, That any active member of the American Medical Association who failed to pay dues for the year 1950, and who was suspended for such delinquency, may be reinstated during the first six months of 1954 by the payment of 1954 dues, only. Should such an individual fail to pay his 1954 dues by July 1, 1954, he shall continue to be considered delinquent.

A study in membership classifications is now under way by the Board of Trustees, it was announced, which should help to eliminate future membership difficulties.

The proposal to extend medical and hospital insurance protection came in the form of a resolution requesting the Council on Medical Service to be ready with definite suggestions for implementing extensions at the 1954 clinical meeting. The resolution called attention to two important groups of citizens who are largely unprotected at the present time. For these, improved coverage could-and should be provided. They are "(a) those individuals who suffer catastrophic or long-continued and highly expensive illness and whose financial resources are not adequate to meet the cost thereof; and (b) those citizens who have retired and are living on small incomes and who are not eligible under presently existing public or private plans."

It was pointed out that the need for such extensions is genuine and also that it represents a soft spot in current plans for insurance protection through which federal subsidies may find a ready entrance.

The delegates also added special emphasis at this session to a standing AMA policy involving all prepaid insurance contracts. The resolution was adopted as an emergency measure, and it specifically condemned all insurance contracts which classify any type of medical service as "hospital service." Pathology, radiology, anesthesiology and physiatry were cited as involved in infractions and violations of this established policy.

In that connection, it should also be noted that recommendations were adopted for careful studies to be made by the Council on Medical Service of all types of union labor medical plans, recognizing both the need for many such plans and the dangers involved in unwarranted extensions of original arrangements and especially emphasizing the fact that medical ethics apply to them with the same force as to any insurance contract.

Ethics Uppermost

Medical ethics came in for discussion and action in many phases and applications. Uppermost in the minds of many delegates were the public charges of unethical practices made against medical men in many popular magazines recently. Their concern and the concern of officers and trustees of the Association was reflected in several resolutions. One of these called upon the Board of Trustees to make a study of the actual extent and seriousness of the unethical conduct alleged in these articles. Another was embodied in an amendment of Chapter IV of the Constitution which was adopted at this meeting pursuant to a request made at the June session. It provides that the Judicial Council may censure or expel service, affiliate and honorary, as well as active members of the association, after due notice, for violations of the principles of medical ethics.

It may be of interest to note here that the resolution introduced by Indiana last June on the subject of federal policies governing the grants-in-aid program for care of crippled children in Indiana has been transmitted to the Department of Health, Education and Welfare where assurances have been given that it will receive prompt and careful attention. The special interest of this resolution, which involves the question of possible charges for those able to pay fees and which applies chiefly to states which do not follow the general grants-in-aid pattern, lies in the fact that it is the one that was hastily and mistakenly siezed upon by *Life* magazine last summer for its ill-natured editorial entitled "Watch It Doc!"

Special emphasis was placed on the principle that physicians may not ethically sell their services or accept a salary for them if they may be

exploited in so doing. Decision as to the propriety of any given salary arrangement must depend, however, upon individual facts and circumstances of the case. In response to many inquiries, the Judicial Council re-emphasized the accepted principle that it is unethical for a physician to have a financial interest in a pharmacy in the area in which he conducts his professional activities and where he profits directly or indirectly from the sale of devices or remedies prescribed for his patients. The principle also applies in the case of rental of space to a pharmacy by a physician if the rental is for a percentage on a sliding scale of income.

Actual revision of the Principles of Medical Ethics is under way by the Judicial Council and the Council on Constitution and By-Laws, of which Dr. Louis A. Buie of Rochester is chairman, but waits upon results of studies and questionnaires now being circulated to the membership. Included in changes now under consideration is the proposal made at the June meeting for a change in recommendations for billing procedures where more than one physician participates in the care of the patient. Certain other preparatory steps have been completed and were approved, also, including a change in format and a change in the title which formerly read "Education Information Not Advertising" to read "The Relationship of the Physician to the Media of Public Information."

Bills Approved

Among bills due to come before Congress in 1954 were several which received official sanction of the delegates at this session. One was the Jenkins-Keogh bill which opposes compulsory social security coverage for self-employed persons. The delegates favored, instead, income tax deductions as proposed in the bill, which will permit such persons to build up their own coverage. Another favors transfer of functions, responsibilities and duties of the Department of the Interior and the Bureau of Indian Affairs as they relate to health to the United States Public Health Service; also authorization of admission to United States Public Health hospitals of persons committed by state courts who are Service beneficiaries or narcotic addicts. Still another strongly favored deduction without limitation of medical and dental expenses for income tax purposes. Finally, the delegates reaffirmed their support of the Bricker Resolution (Senate J. Res. 1), limiting treaty-making powers of the President.

Among resolutions presented for the first time at this meeting were several of general interest and importance. One of them called upon the Board of Trustees to make a detailed study of educational standards and requirements of osteopathic, chiropractic and naturopathic schools. This one was adopted and referred to the Board.

Another asked each constituent state association to create its own Committee on Rural Health. A resolution was also adopted asking that all state and territorial medical bodies get in touch with the President's Commission on Intergovernmental Relations which will soon examine the entire grants-in-aid program and offer assistance.

Three resolutions from groups of orthopedists asking that the word "Rehabilitation" be dropped from the title of the Council on Physical Medicine and Rehabilitation were referred to the Board of Trustees for study and a report in June, 1954. The contention of the orthopedists is that rehabilitation is an essential part of their professional function while the function of the Council is largely the investigation of appliances.

Three resolutions, all from Rhode Island, asking that the current "matching plan for interns" in which the Council on Medical Education and Hospitals now participates, be abandoned and that steps be taken to insure a more equitable distribution of interns to smaller hospitals, were not adopted. Extensive committee hearings did not disclose any evidence that the matching plan was depriving small hospitals of interns, and a committee has already been set up, the delegates were informed, to study the whole problem of intern procurement.

In that connection, it should be noted that a resolution asking that the current practice of listing acceptable hospitals, which subscribe to the matching plan, separately from the acceptable hospitals which do not subscribe to the plan, was not adopted either. Instead, the reference committee suggested that the Council prepare one additional list in which non-subscribers are integrated with subscribers. Still another resolution asking that teaching care in veterans' hospitals be given only to veterans with service-connected disabilities failed of adoption, as did a resolution asking that oral prescriptions be permitted for codeine preparations which have little or no habit-forming effect. The latter was refused because of the possibility that usable narcotics might be extracted from large amounts of these preparations.

Malpractice Study Requested

The Board of Trustees was instructed to make a special study of the malpractice insurance situation all over the country, looking to eventual adoption of measures to make certain that this essential type of protection will continue to be available.

Income of the American Medical Education Foundation now totals $1,000,174.00, the delegates were told. This total represents all contributions since the Foundation was started in 1950. Total contributions are now 200 per cent of the total of 1952 and contributions to it from doctors, alone, are 50 per cent greater than in 1952. In addition, the doctors gave $782,885 direct to sixty-five of the seventy-nine medical schools of the United States (the remaining fourteen failed to make a report).

This last amount was given by 22,273 individual contributors. Among other individual contributions mentioned to the delegates was a gift of $2,000 from the American College of Radiology; also the $10,000 a year contributed for the last three years by the Woman's Auxiliary to the American Medical Association. An additional $22,630 has been contributed by state auxiliaries.

Dr. Louis H. Bauer, immediate past president of the American Medical Association, has succeeded the late Dr. Elmer L. Henderson to the post of director of the Foundation. Dr. Edward L. Turner, secretary of the Council on Medical Education and Hospitals, has been designated secretary-treasurer in place of Dr. Donald G. Anderson who resigned recently to become dean of the University of Rochester School of Medicine. Dr. Anderson will continue on the board of directors.

Report from WMA

Drs. Gunnar Gundersen of Wisconsin and Edwin S. Hamilton of Illinois, both trustees of the American Medical Association, were the American delegates to the World Medical Association meeting at the Hague, September 1 to 6, 1953. They reported that an attempt on the part of various government and judicial agencies to draft an international code of medical law had met with vigorous opposition from the doctors' session. The WMA declared officially that only medical men are qualified to adopt a code for medicine; that such a code must be based upon medical ethics and that the WMA, itself, has already adopted its own international code of medical ethics which, incidentally, has been approved by the House of Delegates of the American Medical Association. A chapter on the doctor's duty to society is now being prepared for addition to that code. WMA served notice that it will never accept a code drawn up by jurists or government agencies. The American Medical Association must continue to support the world organization, the delegates were told, because it is the only international body which can speak for the practicing physicians of the world. Dr. Bauer was re-elected secretary-general of the association, with Dr. Austin Smith of Chicago taking the place of Dr. Henderson on the Council, and Dr. Gundersen replacing the late Dr. R. L. Sensenich. The editorial board of the association's *Bulletin* includes Doctors Paul Cibrie, secretary of the French Medical Association; Hugh Clegg, editor of the British Medical Journal; Lorenzo Garcia-Tarnel, secretary of the Spanish Medical Association, and Dr. Smith, who acts as executive editor of the publication. It is of interest to note that the first World Conference on Medical Education met in London in advance of this meeting. Dr. Victor Anderson, of Rochester, is vice president of this conference and attended the meeting.

The report of the general manager, Dr. George Lull, showed a membership of 117,063 in the Association, representing an increase of 7,323 over 1952, according to the reference committee on officer reports, and showing the continued confidence of the profession in the American Medical Association. All nine professional publications of the Association show an increase in circulation, including the *Journal of the American Medical Association*, which currently circulates 167,000 copies weekly. *Today's Health*, on the other hand, circulates 260,000 monthly, of which only 26,000 go to doctor-subscribers, and it shows a deficit of $150,000 for last year. There is a serious question whether publication of *Today's Health* can be continued but it was noted, also, that if doctor-subscribers could be increased to 100,000, the deficit could be erased.

The delegates heard from the new president, Dr. Edward J. McCormick, and from Mrs. Leo J. Schaefer, president of the Woman's Auxiliary to the American Medical Association. Dr. Chester S. Keefer, newly appointed secretary for health in the Department of Health, Education and Welfare, was there, also, to make his first appearance before the delegates, and Dr. Leslie M. Fitzgerald, president of the American Dental Association, was among a group of distinguished guests who brought greetings from other organizations.

The address of the president stressed some very real social and economic problems of the profession.

Public Relations Discussed

The public relations of medicine in America today are not good, Dr. McCormick said, and expensive public relations programs are not going to do much to improve them. State and county medical organizations are the ones that will have to do the work, he declared, and he urged every delegate to go home and campaign for strong grievance committees and the prompt expulsion of all who disregard the principles of medical ethics.

"This is a grass-roots problem," Dr. McCormick said. "Good public relations cannot be bought. They must be earned by exemplary conduct and genuine service in the public interest.

"We have a policing job to do. Our excellent work in all branches of medicine and the public service are easily forgotten. But not unethical practices of any kind. What we can do is to seek perfection always. In so doing we shall find good public relations. There is no other answer."

Dr. McCormick warned the delegates, also, that the greatest battle to preserve our American system of medicine is yet to come. Already this battle is shaping up in demands for medical care of veterans; in new demands for medical care plans by union labor; in the current system of drafting doctors to care for dependents of service men; in the growing demands for federal subsidies to pay for voluntary insurance. He sees it in statements

of elected officials who still believe it is good politics to encourage federal handouts of all kinds and who "by inuendo and by word, magnify the medical needs of the people and pay little heed to the many more important problems affecting American men and women."

Since American medicine is actually the best in the world, Dr. McCormick suggested to popular writers and orators that they devote more of their attention to the far greater problems affecting the health and welfare of the people, such as housing, sanitation, juvenile delinquency, nutrition, un-Americanism in many institutions, dishonesty among public officials, gambling, gangsterism and other cancers on our national life "including the collectivists, socialists, communists and those who, having lost sight of God, have forgotten that we are a great nation because the Constitution was written by men of deep faith in the Supreme Being, faith in opportunity, faith in private enterprise."

Dr. Keefer declared that the major question before the profession is this:

"How can we continue to produce and promote the finest medical care in the world today?

"Our needs," he said, "are for improved medical education; for co-ordination and extension of facilities and personnel; for geographical extension of services and for bringing the cost within the reach of more people.

"These needs will be met in our free society by the combined effort of the laymen, and of members of the medical and health professions working closely together with a sympathetic understanding in their local communities.

"We need experimentation," Dr. Keefer declared further, "in order to find the answers to many of our problems. Experimentation should be carried out by the profession with the full co-operation of the people of the community and the maximum decision should be made at the community level."

Meetings Well Attended

The Council on Scientific Assembly reported plans well developed for the next annual session in San Francisco, June 21 to 25, 1954, and the next clinical meeting scheduled for December in Miami. It was noted that the winter meetings have been well attended, and the importance of these, as well as the annual meetings, has prompted a consolidation and reorganization of the council activities. A plan was approved for transferring the council from its status as a committee of the House of Delegates to a committee of the Board of Trustees with a permanent office at Association headquarters in Chicago. The council was enlarged and the Bureau of Exhibits was incorporated into its structure.

One of the important events of the winter meeting is the selection of the recipients for the annual General Practitioner of the Year award by the Board of Trustees. The choice this year was Dr. Joseph I. Greenwell, an eighty-year-old active practitioner from New Haven, Kentucky, who has practiced for the last fifty-three years within a 25-mile radius of New Haven. Dr. Greenwell has delivered almost 4,300 babies and, of these, 80 were born last year. He has served as a councilor from the fourth district of the Kentucky State Medical Association for thirty years and is a past president of the Nelson County Medical Society. He is also acting health officer for Nelson County and consulting physician for the Louisville and Nashville Railroad and Gethsemane Abbey. Of his twelve children, eight are living. Two were lost in World War II.

Several Minnesota physicians played an important part in the St. Louis session. Among them were Drs. William L. Benedict, of Rochester, who is also chairman of the Section on Ophthalmology, Dr. Frank J. Elias, of Duluth, and Dr. O. J. Campbell, of Minneapolis, all of whom served on reference committees.

J. A. Bargen, M.D. George Earl, M.D.
O. J. Campbell, M.D. F. J. Elias, M.D.
Delegates to the American Medical Association

MATERNAL MORTALITY SURVEYS

(Continued from Page 144)

sociation in co-operation with the Minnesota Department of Health. Also co-operating in the study were the Department of Obstetrics of the University of Minnesota and the Minnesota Hospital Association. The maternal mortality rate was 2.0 per 1,000 live births and the report concluded that three-fourths of the deaths were preventable. The Committee's report for 1951 shows that the rate has declined to 0.4 per 1,000 live births and that only about one-third of the deaths studied were considered preventable.

Editorial comment on Minnesota's maternal mortality surveys in the January 1953 issue of the *American Journal of Public Health* is of interest. "In only a sixth of all the states is there a concerted and continuing effort to improve maternal mortality experience through investigation, reporting, and consultation. Minnesota has succeeded in reducing maternal deaths from 2 to 0.6 per 1,000 births (1950)—in one decade—and aims to do still better. Does this story suggest anything to the other five-sixths of the states?"

A. B. Rosenfield, M.D., M.P.H.

◆ Reports and Announcements ◆

AMERICAN COLLEGE OF SURGEONS

A four-day Sectional Meeting of the American College of Surgeons featuring clinics and specialty programs will be held at the Hotel Fontenelle in Omaha, Nebraska, March 1-4, 1954. The medical faculties of Creighton University, the University of Nebraska, and seven local hospitals are co-operating in preparations for the meeting.

Eminent scientists and clinicians from many medical centers will participate in the program which will be of unusual and timely interest.

Morning clinics will be held at the teaching hospitals and at the hotel there will be special sessions in general surgery, ophthalmology, obstetrics and gynecology, otolaryngology, orthopedic surgery, pediatric surgery, thoracic surgery, and urology.

All members of the medical profession are invited to attend. Fellows of the College, members of the Junior Candidate Group, interns and residents are admitted without registration fees. Other doctors are charged a $5.00 fee, all of which goes toward paying a portion of the expenses incurred by visiting speakers.

SYMPOSIUM ON RECENT ADVANCES IN THE STUDY OF VENEREAL DISEASES

The Sixth Annual Symposium on Recent Advances in the Study of Venereal Diseases will be held in the auditorium of the Department of Health, Education, and Welfare, Washington, D. C., April 29 and 30, 1954, according to Dr. James K. Shafer, Chief of the Public Health Service's Division of Venereal Disease.

The sessions are open to all physicians and workers in allied professions who are interested in participating These symposia usually draw hundreds from all parts of the country and are the occasion for exchange of the latest available information by some of the outstanding authorities in the field of venereal disease. Topics discussed at this symposium will cover many aspects of venereal disease control including basic and clinical research, serology, epidemiology, treatment, program operation, and professional education.

AMERICAN HEARING SOCIETY'S ANNUAL COMPETITION KENFIELD MEMORIAL AWARD

Competition for the Kenfield Memorial Scholarship, awarded annually by the American Hearing Society to a prospective teacher of lip reading, will open March 1. Application blanks may be obtained by writing to the society's national headquarters, 817 14th St. N.W., Washington 5, D. C.

Deadline for returning completed applications is May 1. They are to be mailed to Mrs. Eleanor C. Ronnei, care of the New York League for the Hard of Hearing, 480 Lexington Ave., New York 17, N. Y. Mrs. Ronnei is chairman of the American Hearing Society's Teachers Committee. The winner will be announced during National Hearing Week, May 2 to 8.

Funds for the scholarship were subscribed in 1937 in memory of Miss Coralie N. Kenfield, San Francisco, California, who was nationally known for her advanced methods in teaching lip reading.

Winner of the annual award is entitled to take a teacher training course in lipreading from any school or university in the United States acceptable to the Teachers Committee. The scholarship is to be used within one year from the date of award.

A satisfactory applicant for the award must be a well-adjusted individual with a pleasing personality, legible lips, good speech and voice, and no unpleasant mannerisms. Graduation from college with a major in education, psychology and/or speech is a requirement.

Specifications for a hard of hearing contestant include thirty clock hours of private instruction in lipreading from an approved teacher or sixty hours of lipreading in public school classes under an approved teacher. Rules for competition state that an applicant shall plan to teach lipreading with or without other types of speech or hearing therapy.

THE ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY—MIDWESTERN SECTION

The annual meeting of the Midwestern Section of the Association for Research in Ophthalmology was held Sunday, February 7, 1954 at the University of Chicago Medical Schools, Chicago.

The program for the meeting included the following papers:

Further Contributions to the Enzymology of Corpus Vitreum. *Albert Zeller,* Northwestern University, Chicago.

Metabolic Studies of Lens Epithelium in Vitro. *Bernard Schwartz,* Betty Danes, and *P. J. Leinfelder.* University of Iowa, Iowa City.

The Liebmann Effect in Binocular Perception. *Maressa Hecht Orzack* and *T. F. Schlagel, Jr.,* Indiana University Medical Center, Indianapolis.

Uveitis. A Preliminary Report. *Howard D. Ostler,* University of Iowa, Iowa City.

Studies in Flicker Fusion of the Eye. *Garth J. Thomas,* University of Chicago.

Variations in Quality of Target for Flicker Fusion Fields. *Claude Trapp* and *Paul W. Miles,* Washington University, Saint Louis.

Some Effects of Yttrium 90 Upon the Posterior Ocular Segment. *Frank W. Newell, Paul V. Harper,* and *Aune M. Koistinen,* University of Chicago.

After-Image Transfer Tests in Anomalous Correspondence. *A. K. Hansen,* University of Iowa, Iowa City.

Preliminary Studies in the Aqueous Following Radiation of the Eye. *David Schoch* and *Irving Puntenney,* Northwestern University, Chicago.

Seton Procedure in Glaucoma. *Edward Pushkin, A. C. Biegel* and *Martha Folk,* University of Illinois, Chicago.

The Electroretinogram in Choroideremia. *George W. Bounds, Jr.,* and *Theodore Johnston,* University of Iowa, Iowa City.

Electroretinography in Artificial Partial Avitaminosis A. *Robert J. Davis,* University of Iowa, Iowa City.

FELLOWS ASSOCIATION OF THE MAYO FOUNDATION

Election at the annual business meeting of the Fellows Association of the Mayo Clinic, held at Plummer Hall, Rochester, December 9, resulted as follows: Dr. H. C. Habein, Jr., president; Dr. John O. Godden, vice president, formerly secretary; Dr. S. E. Sivertson, secretary; Dr. Edward B. Waldman, treasurer.

GP ACADEMY ANNOUNCES OUTSTANDING PROGRAM

Attention of general practitioners is called to the Sixth Annual Scientific Assembly of the American Academy of General Practice to be held in Cleveland, Ohio, March 22 to 25, 1954.

A lectureship and exhibit program designed especially to give the general practitioner diagnostic techniques and procedures which apply to everyday practice has been arranged for this unique three-day session.

Speakers, who are top authorities in their fields, include among others, Sir Alexander Fleming of London, discoverer of penicillin; Dr. E. J. McCormick, president of the American Medical Association; Dr. Howard Rusk, outstanding authority on rehabilitation procedures; Doctors George Crile, Jr., and George H. Curtis of the Cleveland Clinic; Dr. Clarence S. Livingood of the Henry Ford Hospital in Detroit; Dr. Sol Katz, associate editor of GP, official publication of the Academy; and Dr. John C. Krantz, Jr., of the University of Baltimore. More than forty-five scientific exhibits will supplement the scientific program which is under the general supervision of Dr. John F. Mosher of Coeymans, New York.

Social events will include a president's reception, music and dancing and a fashion show, coffee hour and book review for visiting women.

BLUE EARTH COUNTY MEDICAL SOCIETY

Election of officers of the Blue Earth County Medical Society was held December 14 at Mankato with the following elected: Dr. R. Wynn Kearney, president; Dr. J. J. Eustermann, vice president; Dr. William S. Chalgren, secretary-treasurer; Dr. L. M. Hammar, delegate to the Minnesota State Medical Association.

FOUR-COUNTY MEDICAL SOCIETY

The Four-County Medical Society and Auxiliary met at the Mayo Foundation House for afternoon and evening sessions, January 6. Talks given in the afternoon were on "Treatment of Hypertension," "Current Use of Antibiotic Drugs," "The Orthopedic Examination of the Back and Hip," with a demonstration, and "The Management of Frequently Encountered Skin Disorders."

After dinner the women toured the new Mayo Clinic building, while the doctors attended a meeting held in conjunction with the regular staff meeting of the Mayo Clinic.

GOODHUE COUNTY MEDICAL SOCIETY

At a dinner meeting held at Red Wing, December 3, the following officers were elected to head the Goodhue County Medical Society: Dr. Ezra Bridge, Mineral Springs Sanatorium, president; Dr. R. V. Sherman, vice president; Dr. George M. B. Hawley, secretary-treasurer.

ST. LOUIS COUNTY MEDICAL SOCIETY

Election of officers of the St. Louis County Medical Society took place at the Kitchi Gammi Club, Duluth, December 11. The following officers were elected: Dr. S. H. Boyer, Jr., installed as 1954 president; Dr. K. R. Fawcett, Duluth, president-elect for 1955; Dr. Walter S. Neff, Virginia, vice president, Virginia; Dr. F. R. Kotchevar, Eveleth, member of the judiciary committee; Dr. Paul Reed, Virginia, member of the economics committee, and Dr. Clarence N. Jacobson, Chisholm, and Dr. Neff, delegates.

STEARNS-BENTON COUNTY MEDICAL SOCIETY

Stearns-Benton Medical Society met at the Granite Bowl, St. Cloud, December 17, electing the following officers: Dr. Henry Reif, St. Cloud, president-elect for 1955; Dr. John Kelly, Cold Spring, president—elected last year; Dr. William Autry, vice president; Dr. E. W. Anderson, secretary.

TRI COUNTY MEDICAL SOCIETY

Doctors and their wives from Kandiyohi, Meeker and Swift Counties met at the annual dinner of the Tri County Medical Society, held in Willmar, December 17. Lakeland Medical Center and Willmar Clinic staffs acted as hosts, with Dr. Roger P. Michels, Willmar, presiding. Musical numbers included one on the violin by Dr. Robert Peterson with Dr. L. J. Opsahl at the piano. Dr. Peterson and Dr. Opshal with Dr. A. M. McCarthy gave a humorous specialty number.

Dr. Paul Giddens, president of Hamline University, spoke on the danger that confronts academic freedoms today, and also pointed out the need for greatly increased facilities in teaching and school rooms for the oncoming tremendous increase in school population.

MINNESOTA STATE BOARD OF HEALTH

Dr. F. W. Behmler, Morris, was re-elected president of the Minnesota State Board of Health at the annual meeting of the board held in Minneapolis, January 12. Prof. Herbert Bosch, University of Minnesota, was named vice president, and Dr. A. A. Chesley was re-elected secretary and executive officer. Dr. Behmler has been a member of the State Board of Health since 1940.

Of General Interest

Dr. John W. Ferree, director of community service and education, New York City, and Rome Betts, executive director of the American Heart Association also of New York, were in Minneapolis December 16 to discuss the 1954 Minnesota Heart Fund program set for February. They conferred with **Dr. W. S. Neff,** Virginia, on community service; with **Dr. Karl Anderson,** Minneapolis, on cardiac problems in industry, and **Dr. John Briggs,** Saint Paul, on professional education. They also met with the State Medical Association's executive committee during the day, and in the evening with the general membership at a meeting at the University of Minnesota.

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Yellow Medicine and Renville Counties qualified for tuberculosis accreditation recently. Attended by approximately ninety persons from the two counties, a banquet was given for the occasion December 7, at the Romberg Hotel, Sacred Heart. To qualify for accreditation a county must have a tuberculosis death rate averaging ten or less per 100,000 population for a five-year period. Yellow Medicine County had a 3.7 and Renville a 5.8 rate for the years 1948 to 1952. The county must also have a low infection rate which is established by sampling high school seniors through a tuberculin testing program.

Dr. M. S. Nelson, serving for many years as president of the Yellow Medicine County Tuberculosis Association; **Dr. Lewis S. Jordan** and **Dr. Kathleen Jordan** of the Riverside Sanatorium, Granite Falls, and Clara Thorpe, the organization's secretary for many years, were presented with framed certificates in recognition for the work they have done in combatting tuberculosis. The awards were made by Marguerite Breen, public relations officer for the Minnesota Tuberculosis and Health Association.

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Dr. Pao-shu Chen, Formosa, and Dr. Andreas Angara, the Philippines, visited the Hennepin County League for Planned Parenthood recently for information they might use to help their countrymen. **Dr. Marian Grimes,** planned parenthood clinician, explained the operations of the clinic, and gave the two doctors data on birth control. Dr. Chen, public health student at the University of Minnesota on a World Health Organization scholarship, said there was a great need of birth control in Formosa, where, though the area is the same size as Maryland and Delaware combined, has a population four times that of the two states. Dr. Angara, studying public health at the University of Minnesota on a year's scholarship from the Foreign Operations Administration, was primarily interested in the "rhythm" method of planned parenthood for the underpopulated Philippines. Both doctors said the greatest problems in health, in their respective countries, were sanitation—water supply and waste disposal.

Dr. John Grant, Sauk Centre surgeon, specializes in "organs" in his spare time. He builds pipe organs, which he says he learned how to build when he read a few books and talked to some "guys in the business" during his medical school days. After the reading and advice from "the guys in the business," he began buying parts and assembling the pipe organs. His last venture is a 1,300-pipe organ at the Church of the Good Samaritan at Sauk Centre which he built with the help of the pastor, Rev. Nor Schoenheider. The organ, built from parts secured from churches in Minneapolis, St. Paul, Excelsior and Willmar, was dedicated December 20.

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Among Fellows of the Mayo Foundation receiving degrees at the Commencement exercises at the University of Minnesota December 10 were the following: **Dr. Robert C. Bahn,** Ph.D. in pathology; **Dr. Homer R. Warner,** Ph.D. in physiology; **Dr. Dean P. Epperson,** Ph.D. in surgery; **Drs. Earl F. Beard, Fred Z. Havens, Jr., Alexander M. Minno, Waldemar N. Person, John D. Ruff** and **Robert G. Tompkins,** M.S. in medicine; **Dr. Gilbert A. Reese,** M.S. in ophthalmology; **Dr. Eran O. Burgert, Jr.,** M.S. in pediatrics; **Dr. Milton E. Kurth,** M.S. in plastic surgery; **Dr. Charles F. Smith,** M.S. in radiology; and **Drs. Mitchell S. Madison, J. D. Mortenson, Harold W. Neuman** and **Donald N. Vivian,** M.S. in surgery.

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Dr. Gordon Uhley, Crookston, is taking a year's leave of absence from the Northwestern Clinic, Crookston, in order to do graduate work in urology with Dr. S. D. Creevy at the University of Minnesota, Dr. Uhley's alma mater.

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Dr. Marie Bepko, Cloquet, has apparently mastered the art of budgeting time. She is physician, partner with her husband, **Dr. R. H. Puumala,** homemaker, and active in several professional and civic organizations.

Born in Chicago of Czechoslovakian parents, Dr. Bepko graduated from the University of Illinois, College of Medicine, and interned at the American Hospital during which time she married Dr. Puumala, who was at this time resident at University of Illinois Research Hospital. After their first child was born Dr. Bepko continued her preparation by taking a six months' residence in psychiatry at the state hospital in Elgin, Ill. The family moved to Cloquet in 1935 where the two doctors have since practiced.

Though both doctors have travelled extensively, they have not taken their vacations together until last summer, when with their two children, they motored to Anchorage, Alaska, via the Alcan highway. Besides her duties to her family, Dr. Bepko is vice president of the Pan American Women's Alliance; life member of the American Medical Wom-

en's Association; president of the Minnesota State Medical Women's Association; active in community activities which include Parent-Teacher work, projects of the Business and Professional Women's Club, and other organizations.

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Dr. J. W. Bratholdt and **Dr. L. J. Larson,** Watertown, moved their offices into the newly constructed Bratholdt Clinic December 12. The clinic is located across the street from the Bratholdt Hospital.

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Dr. Harold M. Solvason, formerly located at Hennepin and Franklin Avenues, Minneapolis, has opened a new office at Fifty-fourth and Nicollet, Minneapolis. Dr. Solvason is a graduate of the University of Minnesota School of Medicine.

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Dr. Francis J. Braceland, Hartford, Connecticut, formerly of the Mayo Clinic, is a member of one of the divisions of the task force on Federal Medical Services of the Hoover Commission. The activities of the division is to gather information from government agencies and other sources before any conclusions are reached or any recommendations given to the Commission.

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Dr. Edward Zupanc, formerly of Granville, Wisconsin, who has served with the Armed Forces for two years in Japan, will open an office in Duluth, specializing in pediatrics.

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Dr. Swan Ericson, Le Sueur, has retired after practicing medicine in the community for the past thirty-one years. Dr. Donald A. Limbeck graduate of the University of Nebraska, Medical School, is taking over Dr. Swanson's practice.

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Dr. Edward Q. Ertel, Ellendale, recently contributed a microscope to the Ellendale High School. The gift included a wood carrying case, glass bell protective cover, and immersion lens.

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Awarded for his work in establishing radiological teaching programs in various medical schools in India, **Dr. Leo G. Rigler,** Minneapolis, received the gold medal of the Radiology Society of North America December 23. Dr. Rigler, head of the University of Minnesota's Radiology Department visited India early last year as a member of the World Health Organization specialists' team.

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Dr. William Feigal, Falls Clinic, Thief River Falls, addressed the monthly meeting of the Roseau County Medical Society, December 11. Together with his subject, "Urology in Northern Minnesota," he also reviewed x-rays, showing pathological findings in this area with their treatment and clinical course.

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Dr. David R. Johnson and **Dr. Lyle A. French,** Minneapolis, spoke on the topic, "Cerebral Hemispherectomy for Intractable Seizures," at the regular meeting of the Minnesota Society of Neurology and Psychiatry, at the Town and Country Club, St. Paul, January 12. **Dr. Frank Kiesler,** also Minneapolis, spoke on "The Patient who Can't be Cured."

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Dr. D. H. Garlock, Bemidji, has recently been certified as a Tree Farmer in the state and national tree farm program. His 155-acre farm east of Lake Bemidji was approved by the tree farm committee of Keep Minnesota Green.

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Dr. F. H. Magney, Duluth, was given the Silver Beaver Award high scouting honor, at the annual business meeting of the North Star Council, Boy Scouts of America, at Duluth Chamber of Commerce quarters December 7. Dr. Magey was cited for long service (since 1935) in keeping the boys fit "in matters pertaining to health."

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Dr. Donald H. Honath, Detroit, Michigan, has joined **Dr. A. J. Olson** and **Dr. F. C. Anderson,** at their clinic in Owatonna. Dr. Honath is a graduate of Wayne University College of Medicine; interned at Evangelical Hospital, Chicago, and finished a four and a half-year residency in general surgery at Mt. Carmel Mercy Hospital, Detroit.

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A new series of industrial safety schools sponsored by the Duluth Chamber of Commerce was held in Duluth, December 9. **Dr. John F. Briggs,** Saint Paul addressed the general session on "Optimism in Heart Disease." **Dr. James Tetlie,** Duluth, spoke on eye injuries; **Dr. Kenneth A. Storsteen,** Duluth, on gastro-intestinal disease; **Dr. Robert J. Goldish,** Duluth, on diabetes; **Dr. John Schmid,** Duluth, on industrial dermatitis; **Dr. Walter Kelley,** on burns; **Dr. William Grohs,** hernias; **Dr. Elizabeth Bagley,** "the home, an important industry," at the women's section.

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Dr. R. J. Ripple, Saint Paul, spoke to members of the Washington County Medical Society at the meeting, December 8, at Stillwater, on the problems of resuscitation and anesthesia. Doctors from Forest Lake and Hastings attended the meeting which was followed by dinner.

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Dr. G. M. Hawley spoke on "Ministering to Hospital Patients" at a meeting of the Red Wing Ministerial Association held December 7, at the Baptist parsonage, Red Wing.

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The new Veterans Research Hospital, Chicago—key hospital in the veterans hospital system—has three Minnesota doctors on its staff. **Dr. Richard V. Ebert,** chief of medicine; **Dr. Ben I. Heller,** chief of the metabolic section; **Dr. Craig Borden,** chief of the cardio-vascular section. All three men have faculty appointments at Northwestern University.

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Dr. F. H. Baumgartner, Albany, has resigned from his office as mayor of Albany. For ten years he has spearheaded drives that have brought about improvements in the village, but his responsibilities as physician has increased to such an extent, he was unable to carry on both tasks.

On January 2, **Dr. K. A. March,** Cambridge, was joined in his practice by Dr. Michael Danyluk, originally from Germany. Dr. Danyluk has been on the staff at the Swedish Hospital, Minneapolis, since coming to Minneapolis in 1950. He studied medicine at the University of Munich, Germany.

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Dr. Albert V. Stoesser, Professor of Pediatrics and Allergy at the University of Minnesota, spoke on "Allergy and Your Child's Behavior" at a meeting of the Child Psychology Study Circle, January 4, at the Groveland Park School Auditorium, St. Paul.

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The complete list of new members from Minnesota accepted by the American College of Surgeons for 1953 is as follows:

Grant L. Griebie, Brownton, surgery; Hubert D. Clapp, Crookston, obstetrics-gynecology; Philip F. Eckman, Duluth, surgery; Francis L. Stutzman, Montevideo surgery; Clarence W. Wasmund, Red Wing, ophthalmology; John W. Kirklin, surgery, Karl A. Lofgren, surgery, and Frank Charles Mann, pathology, all of Rochester; Hanns C. Schwyzer, Saint Paul, surgery, and Jack R. Pierce, Virginia, obstetrics-gynecology; Ivan D. Baronofsky, surgery; Walter P. Eder, surgery; Milton Feinberg, surgery; Davitt A. Felder, surgery; Geddes B. Flagg, ophthalmology; William Harold Ford, surgery; George L. Garske, urology; John K. Grotting, plastic surgery; Carter W. Howell surgery; Arthur W. Ide, Jr., surgery; C. Walton Lillehei, surgery; and Henry A. Norum, surgery, all of Minneapolis.

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Dr. Ernest O. Hanson, who has been practicing in Carlton, has opened his office in Cloquet recently.

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The Hayfield Medical Center, a $10,000 community project, was ready for occupancy January 15, by **Dr. Harold J. Elliot,** former missionary doctor in Africa, who came to Hayfield last fall.

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Dr. J. E. Ponterio, Shakopee, recently had $140 stolen from his cash box which was kept in his office desk. Filing cabinets and desk drawers had been rifled.

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Dr. Henry G. Moehring, radiologist of the Duluth Clinic, Duluth, is one of the co-authors of a three-volume book entitled "Clinical Roentgenology," published by Charles C Thomas, Springfield, Illinois. The first volume is now being offered for sale.

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Progress is being made on the new Adams Clinic, Chisholm, which will be ready for use sometime in February. **Dr. Clarence Jacobson, Dr. Bayerd French,** and **Dr. George Erickson** will have their offices in the building plus rooms and facilities for x-ray and laboratory work.

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More than 1,050 doctors attended the twelfth annual meeting of the American Academy of Dermatology and Syphilology at the Palmer House, Chicago, recently. Among officers elected was **Dr. John F. Madden,** Saint Paul, elected vice president.

Dr. Solveig Gislason, New Ulm, recently accepted a position on the medical staff at the State Hospital, St. Peter. Dr. Gislason, born in a log cabin near Gardae, North Dakota, attended North Dakota schools—Normal School, Valley City, and University of North Dakota, Grand Forks, and then took a claim in Montana. She lived alone on the ranch riding horseback to the school where she was teaching. Interest in further education sent her to University of Minnesota where she graduated from the Medical School in 1921.

Dr. Gislason has written children's books that have been recognized and displayed at fairs and seminars by educational authorities in foreign countries such as Belgium, India, et cetera. She has operated an antique shop in New Ulm, and has also been active in civic and social affairs, especially as president of the New Uum Recreation Commission.

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Appreciated by the communities he served so long, **Dr. Mose Lane Strathern,** Gilbert, still makes his rounds though he began his practice on the range in 1911. He moved to Coleraine with his bride that year to take up practice at $100 a month; later he moved to Gilbert, which was then one of the boomingest towns on the Range. During the horse and buggy days, the fee for delivering babies was $5, when the fee went up to $15, Dr. Strathern bought an automobile. Last October 22, he with nine other members of the team that played Michigan a six to six game in 1904, had a reunion in Minneapolis. Dr. Strathern was captain of the Minnesota team that year.

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Dr. Alan Challman, of Minneapolis, attended the annual meeting of the Advisory Committee to the Psychiatry and Neurology Division of the United States Veterans Administration held in Washington, D. C., January 18 and 19, 1954.

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From a reader: In a recent issue (November) of your excellent magazine, you announced in your news column, the *three* young physicians named by *Time* magazine as "Newsmakers of Tomorrow!"

You neglected to name **Edmund B. Flink, M.D.,** director of Internal Medicine at Veterans Hospital, and member of the University of Minnesota Medical staff. His recognition was as deserving as the other gentlemen. (*We regret the omission.*—THE EDITORS.)

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Dr. George D. Haggard, Minneapolis, observed his ninety-seventh birthday January 18. At this time he also received a Life Membership Certificate from the Minnesota Medical Association of which he is justly proud. He has been interested in the Hennepin County Medical Society over the years, and to use his own words, he said, "Why I can recall years ago when I was secretary of the county society, we were gratified that we could boast of eighty members—and now the county society has well over 1,000 members."

Dr. Haggard retired completely from his practice only two or three years ago. He is making his home now with his daughter.

HOSPITAL NEWS

The St. Cloud Hospital has named the following medical staff for the coming year: **Dr. Otto Phares,** president; **Dr. E. M. Anderson,** president-elect; **Dr. J. B. Gaida,** vice president; **Dr. Vernon Neils,** secretary. Serving on the executive committee with the new officers are: **Dr. L. M. Evans, Dr. L. A. Veranth** and **Dr. Karl Walfred,** past president. Chiefs of services include: **Dr. H. E. Sisk,** medicine; **Dr. C. F. Brigham, Jr.,** surgery; **Dr. R. N. Jones,** anesthesia; **Dr. F. J. Schatz,** obstetrics; **Dr. C. S. Donaldson,** orthopedics; **Dr. W. T. Wenner,** EENT; **Dr. H. J. Reif,** pediatrics; **Dr. P. E. Stangl,** clinical and pathological laboratories; and **Dr. C. B. Nessa,** chief of radiology.

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The Duluth, Missaba and Iron Range Railway made a contribution of $300,000 toward construction of a new hospital at Two Harbors. The money is to be used by the North Shore Memorial Hospital, Incorporated, which is a non-profit group, organized under Minnesota laws, and backed by the Two Harbors Independent Citizens Committee. **Dr. Allen Gunn-Smith,** spokesman for the hospital indicated the group planned the construction of a fifty-bed hospital that would serve the entire North Shore area.

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Fairmont Community Hospital has the following new officers on the medical staff: **Dr. E. E. Zemke,** president; **Dr. O. E. Wandke,** vice president, and **Dr. Neil Wickerson,** secretary-treasurer.

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The annual meeting of the medical staff of St. John's Hospital, Saint Paul, named **Dr. T. S. McClanahan,** chief of staff, December 15; **Dr. Robert W. Emmons** was named first vice chief; **Dr. P. G. Polski,** second vice chief; **Dr. Jack W. Strand,** secretary-treasurer; **Dr. C. Kenneth Cook,** retiring chief of staff, was named to the executive committee for a three-year term along with **Dr. H. L. Stolpestad.**

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The need for a community hospital in Forest Lake is great, according to a recent editorial in *Forest Lake Times.* The need is also great for public spirited people living in the community to attempt the project, while the need for federal aid is also great. The area designated four years ago as one of the high-priority areas for a thirty-five-bed general hospital. **Dr. Helen L. Knudson,** State Department of Health, indicates that she has been unsuccessful finding people interested in the hospital program.

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Tentative plans for the Douglas County Hospital, Alexandria, were shown by Architect Frank Jackson at a meeting of the Advisory Committee recently. Plans, that call for a new hospital architecture, consists of diagonal wings extending Y-like arms from each corner of the one-story building. The hospital, capacity forty-eight beds, will be located on a 17-acre plot of ground in the southeast section of the city. **Dr. L. F. Wasson,** Alexandria, and **Dr. E. E. Emerson,** Osakis, are among the committee members.

Plans for a fund-raising project to finance building a community hospital at Cloquet was approved at a joint meeting of the Cloquet Civic Affairs Council and the rural-urban committee of the Cloquet Chamber of Commerce recently. A poll was taken by postcard which indicated that 97.3 per cent of the people in the community were in favor of the project.

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New officers of the medical staff of Glenwood Hills Hospital were elected at the annual meeting held December 16, 1953, as follows: **Dr. J. A. Resch,** president; **Dr. John Schumacher,** vice president, and **Dr. Irving Bernstein,** secretary.

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MINNESOTA BLUE CROSS-BLUE SHIELD

At its meeting on December 30, 1953, the Board of Directors of Blue Shield accepted the resignation of Arthur M. Calvin as Executive Director effective as of January 1, 1954. In view of the fact that the increased duties and responsibilities which Mr. Calvin's recent election to the office of President of Blue Cross have imposed upon him may prove too great for his continued active leadership in the executive management of Blue Shield, the Board of Directors after considerable deliberation accepted Mr. Calvin's resignation. In so doing, the members of the Board highly commended Mr. Calvin for his distinctive contribution to the successful organization and growth of Blue Shield. As Executive Director from the inception of Blue Shield six years ago, he has seen it grow to cover 600,000 Minnesotans, and its payments to doctors for services rendered subscribers now exceed $4,000,000 annually.

The Board of Directors, having accepted Mr. Calvin's resignation, appointed Dr. Edwin J. Simons as Executive Director. Dr. Simons will continue the executive management of Minnesota Blue Shield, which is now one of the twelve largest Blue Shield Plans in the nation. Dr. Simons was formerly engaged in the practice of medicine at Swanville, Minnesota, for a period of twenty-five years. For over three years, he has served as Medical Director of Blue Shield, and in the past year, he has also served as its Associate Executive Director.

Blue Shield and Blue Cross will continue to co-ordinate their activities as they have in the past. Blue Cross will continue to provide Blue Shield with such services as enrollment, billing, accounting and statistical services. The two organizations will remain in close co-operation to promote the development of both plans in order that the people of Minnesota may be provided with the best in medical-hospital service and coverage.

One person out of every six covered by Minnesota Blue Cross used hospital care during 1953, an increase over the care used in 1952.

Approximately 942,000 days of hospital care were provided by Blue Cross, an increase of about 40,000 days over 1952. This increase in hospital utilization also necessitated an increase in Blue Cross funds paid out. Almost fifteen million dollars was paid out for Blue Cross subscribers and their families in 1953 compared with payments of $12,500,000 in 1952.